MAY 2 - 1934

The Seventeenth Annual Meeting of the American Association for Thoracic Surgery, Boston, Mass., May 31, June 1 and 2, 1934.

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Vol. 3 APRIL, 1934 No. 4

The Journal of Thoracic Surgery

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The American Association for Thoracic Surgery

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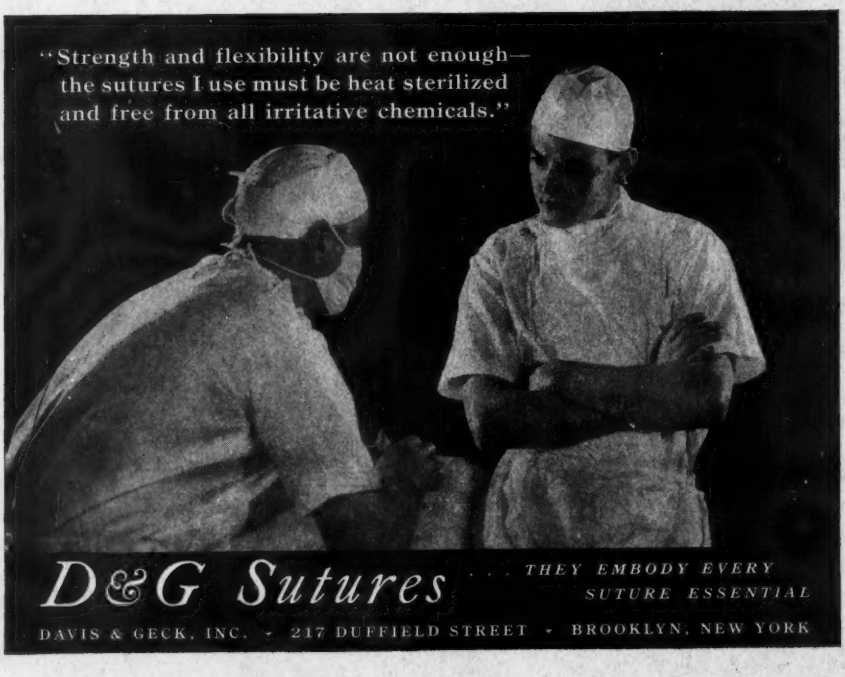
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The Journal of Thoracic Surgery

VOL. 3 APRIL, 1934 NO. 4

Original Communications

THE REINFLATION OF ATELECTATIC LUNG

AN EXPERIMENTAL STUDY*†

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BOSTON, MASS.

THE production of an atelectatic state in the lung by bronchial obstruction was first demonstrated experimentally by Mendelsohn in 1845, and Lichtheim in 1878.[1] Following these publications little appeared in the literature on this subject until after the clinical report in 1908 of Pasteur,[2] who described massive collapse in patients dying with respiratory paralysis complicating diphtheria. Since the World War, numerous experimental and clinical studies on atelectasis have appeared. The causal relation between bronchial obstruction and the subsequent atelectatic state is now generally accepted. The clinical and roentgenographic manifestations have been adequately described by Jackson and Lee,[3] Manges,[4] Holmes,[5] and others.

In the prophylaxis and therapy of the atelectatic state, hyperventilation of the lungs, induced by the breathing of carbon dioxide mixtures, has been advocated by Scott and Cutler[6] and Henderson and his collaborators.[7] This method has had a widespread clinical application. Its efficacy has been questioned recently by King[8] in a large and adequately controlled series of postoperative patients.

The use of positive pressure breathing for the relief of lobar atelectasis has been recommended by Middeldorpf[9] and is being used in some German clinics. Jackson and Lee[3] first proposed and employed bronchoscopic aspiration of obstructing mucous plugs in postoperative massive

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collapse. Yet Jackson states, "A lung collapsed because of a plugged bronchus does not immediately re-expand after removal of the plug. Why, we do not know." Reinflation of atelectatic lung tissue must require that the cohesion of alveolar membranes lying in intimate contact (as the definition of atelectasis implies) be overcome. The adhesive effect of any exudate present, and the resistance to expansion offered by the natural elastic forces of the lung parenchyma, are also forces acting against reinflation. No study in the literature has come to our attention which undertakes to measure the subatmospheric (negative) pressure that must be applied to the pleural surface of a collapsed lobe to effect its reinflation. Neither have any studies been observed on the effective differential positive pressure which must be exerted on the bronchial lumen of a collapsed lobe to effect its reinflation.

It is the purpose of this study to measure these pressures in four species, both in acutely and in chronically atelectatic lungs, and following the administration of inhalation (ether) and parenteral (nembutal) anesthesia.

I. REINFLATION OF AN ACUTELY ATELECTATIC, UNINFLAMED LUNG LOBE IN VIVO AND IN VITRO BY THE DIRECT BRONCHIAL ROUTE

Methods and Materials.—Experiments were conducted upon cats, rabbits, and dogs. The animals were anesthetized with nembutal, given intraperitoneally in doses of from 40 to 45 mg. per kilogram of body weight, or with ether. When ether was used, the induction was by the open cone method, and was followed by the intratracheal insufflation of air-ether vapor from a Becker machine.

When the animal was anesthetized, a tracheotomy was performed and a glass cannula of approximately the same caliber as the trachea was inserted. The tracheal cannula was equipped with a side arm, to which was connected positive pressure insufflation for use when the chest was opened.

Through a right or left intercostal incision in the fifth or sixth space, the chest was opened, and the bronchus to the lower lobe was carefully cleared by blunt dissection. A long and slender glass tube was then introduced through the tracheal cannula down into the lower lobe bronchus, which was tied upon it. Thus the lower lobe was isolated from the remainder of the lung field. With the lungs well expanded, the chest was closed in layers, and the positive pressure respiration was discontinued. In the case of ether anesthesia, however, insufflation was continued at a lower pressure level (1 or 2 cm. of water).

The isolated lower lobe was then irrigated through the glass tube with small quantities (25 to 50 c.c.) of pure oxygen, the lobe partly deflated by aspiration, and the glass tube closed. At the end of from one and one-half to three hours the chest wound was reopened under positive

pressure respiration; the lower lobe was always found completely atelectatic and liverlike in its color.

In some experiments the reinflation was conducted in vitro. The atelectatic lobe was rapidly excised and suspended in a respirator. The respirator was a wide-mouthed glass jar closed by a tightly fitting rubber stopper in which were four openings. Three openings carried short glass cannulas and the fourth accommodated a thermometer, all tightly fitted. The inner end of one glass cannula was tied into the main bronchus of the atelectatic lobe, while its outer end was free to the atmosphere. The outer end of the second glass cannula was connected to a water manometer, and the third to an air-suction line. The respirator was placed in a water-bath to keep the temperature between 95° and 100° F.

Beginning with –2 cm. of water, the pressure in the chamber was alternately lowered and returned to atmospheric at a rate of from 14 to 18 per minute. Every five minutes the lower limit of the pressure was extended by an additional 2 cm., and the experiment was continued until the lobe was reinflated completely. The pressure level at which the first signs of alveolar inflation were seen and the level at which the lobe was completely reinflated were recorded. These values are termed the initial and final pressures respectively.

TABLE I

MINIMAL PRESSURE REQUIREMENTS FOR REINFLATION OF ACUTELY ATELECTATIC LUNG

ANIMAL	EXP.	TYPE OF PRESSURE	TYPE OF ANESTHESIA	PRESSURES, CM. OF WATER	
				INITIAL	FINAL
Cat	9	negative	nembutal	–16	–18
	11	negative	nembutal	–14	–17
	12	negative	nembutal	–16	–22
	14A	negative	nembutal	–14	–22
	35	negative	nembutal	–12	–16
	24	positive	nembutal	+12	+16
	25	positive	nembutal	+12	+16
	26	positive	nembutal	+14	+16
	27	positive	nembutal	+14	+16
Rabbit	1	negative	nembutal	–14	–18
	2	negative	nembutal	–16	–20
Dog	6	negative	ether	–14	–16
			Mean:	±14	±18

In other experiments the reinflation was conducted in vivo with positive air pressure applied to the bronchial lumen. The chest wound was held retracted so that a continuous inspection of the atelectatic lobe could be made. The tube supplying the occluded lobe was connected with a source of controlled positive air pressure, and the lobe was insufflated with air, beginning at +2 cm. of water and alternating to atmospheric pressure 14 to 18 times per minute. The pressure was increased +2 cm. of water every five minutes until reinflation was complete. The pressure

at which alveolar inflation began and the pressure at completion of inflation were recorded. These two values were termed the initial and final pressures respectively.

Results.—Acute experiments were performed on 9 cats, 2 rabbits, and 1 dog. The results of this series are presented in Table I.

The results show that the minimal pressures necessary to initiate reinflation were within narrow limits identical for individual animals and for the three species studied. Moreover, as would be expected, the effective pressure was the same whether positive pressure (intrabronchial insufflation) or negative pressure (extrapulmonary) was used.

The rather constant level at which reinflation began indicated that a critical threshold pressure might exist. The following protocols are from experiments designed to test this assumption.

Protocol 1.—Cat No. 27. Weight 4.35 kg.

10:00 A.M.—170 milligrams of nembutal injected intraperitoneally.
10:30 A.M.—Tracheotomy; insertion of tracheal cannula. Thoracotomy in right fifth interspace; lobe cannula tied into right lower lobe bronchus.
10:50 A.M.—Chest closed in layers.
10:55 A.M.—Occluded lobe irrigated with oxygen; deflated; cannula occluded.
12:25 P.M.—Chest wound reopened under positive pressure. Lower lobe solidly atelectatic; its cannula connected to insufflation apparatus.
12:30 P.M.—Insufflation of lower lobe bronchus started at +9, alternating to +4, 20 times per minute. Animal kept on heating pad and covered with blankets; warm saline compresses to thoracotomy wound.
2:30 P.M.—Additional nembutal, 20 mg.
4:30 P.M.—Additional nembutal, 20 mg.
6:35 P.M.—No visible reinflation of lower lobe.
6:36 P.M.—Pressure raised to +14; reinflation began actively.
6:40 P.M.—Reinflation completed at +16 cm.

Protocol 2.—Cat No. 26. Weight 3.5 kg.

10:30 A.M.—140 milligrams of nembutal injected intraperitoneally.
10:45 A.M.—Tracheotomy. Under positive pressure a right thoracotomy was performed; lobe cannula introduced through tracheal cannula and tied into lower lobe bronchus.
11:25 A.M.—Chest closed in layers.
11:35 A.M.—Occluded lower lobe irrigated with oxygen in from 25 to 50 c.c. quantities; deflated; cannula closed.
1:12 P.M.—Chest wound reopened; margins retracted. The lower lobe appeared completely atelectatic. Lobe cannula was connected to positive pressure and insufflated to +9 or +10 cm. H_2O at a rate of 18 per minute.
3:30 P.M.—Additional nembutal, 25 mg.
5:15 P.M.—No demonstrable reinflation of the lobe.
5:16 P.M.—Pressure raised to +14; immediate reinflation, completed at +16 cm.

These experiments clearly demonstrated that there is a threshold pressure which must be reached before reinflation begins at all; in other words, prolonged application of a subthreshold pressure has no cumulative effect.

II. REINFLATION OF AN ACUTELY ATELECTATIC, UNINFLAMED LUNG BY THE COLLATERAL ROUTE

The work of Van Allen and Soo[10] has shown that an atelectatic lung lobule may become reinflated spontaneously by the passage of air collaterally from adjacent, freely ventilated lobules when its own direct bronchial communication remains obstructed.

The pressure required for the collateral reinflation of an acutely atelectatic lung lobe as compared with that required for the direct reinflation of such a lung lobe by the normal bronchial route was determined in the following experiment.

Protocol 3.—Cat No. 35. Weight 3.2 kg.

12:00 A.M.—130 milligrams of nembutal injected intraperitoneally.

1:00 P.M.—20 milligrams of nembutal additional.

2:00 P.M.—Tracheotomy. Insertion of tracheal cannula. Under positive pressure the left chest was opened through an intercostal incision, the lower lobe bronchus freed up and cannulated. The left lower lobe was then irrigated with 50 c.c. portions of oxygen, deflated, the bronchus tied off, and the cannula removed.

2:30 P.M.—Left chest wound closed. The same procedure was repeated on the right side.

2:50 P.M.—Operation completed.

3:35 P.M.—Chest opened. Both lower lobes were seen to be completely atelectatic in appearance. They were excised. The right lobe was cannulated in the usual manner. On the left, the first side bronchus was carefully freed up at the hilus and cannulated. The main bronchus was ligated below this first side branch, so that the latter offered the only route for access of air. The two lobes were then placed in the same respirator system, and reinflation studies carried out as described under *Methods*.

TABLE II

ELAPSED TIME IN MINUTES	PRESSURE CM. WATER	RIGHT LOBE (DIRECT REINFLATION)	LEFT LOBE (COLLATERAL REINFLATION)
0	− 8	None.	None.
5	−10	None.	None.
10	−12	Began in one lobule.	Began in the cannulated lobule.
15	−14	Many focal areas of reinflation.	Slow reinflation by an advancing margin from the cannulated lobule.
20	−16	Complete.	Many focal areas of reinflation.
25	−18		Complete.

This experiment was repeated in a dog with similar results. It was observed that there was no essential difference in the pressure threshold by direct or collateral routes.

III. REINFLATION OF A CHRONICALLY ATELECTATIC, UNINFLAMED LUNG: ETHER AND NEMBUTAL ANESTHESIA

Materials and Methods.—Cats were used exclusively. Anesthesia was induced by nembutal or ether, as in the preceding section. The right chest was prepared by shaving and painting with iodin and alcohol. A sterile catheter was inserted into the trachea with the aid of a laryngoscope, and rhythmic positive pressure air insufflation was begun. The chest was opened in the fifth or sixth interspace under aseptic technic: the bronchi of the right lower lobe and accessory lobe were ligated with No. 14 silk, either separately or together at their junction. In one group of animals a second ligature was passed loosely about the primary bronchus just above the middle lobe branch; this ligature was simply looped about the bronchus without occluding it, in order to permit the easy identification of that bronchus and redissection at a subsequent operation. In all the cats the unobstructed lobes were fully inflated and the chest was closed in layers with silk. The animal was allowed to recover; was kept in a cage on a standard animal house diet until required. The duration of the atelectasis was computed from the day of operation, since it is known to develop within twenty-four hours following ligation of a bronchus.

On the date selected for the reinflation experiment the animal was given nembutal intraperitoneally, the chest wound was reopened and inspected. The atelectatic lower lobe was excised, any stringy mucus present in the bronchus was aspirated, and reinflation carried out in the respirator, as described in Part I. In those animals in which the reinflation was carried out by positive pressure insufflation in vivo a tracheotomy was performed and the usual large glass tracheal cannula was inserted. Under positive pressure respiration the chest wound was reopened; the guide ligature on the primary bronchus was picked up, and the bronchus redissected from its adhesions. A slender cannula was passed through the trachea and tied into the primary bronchus. Having by this maneuver separated the operative atelectatic lobes from the others, the ligature occluding the lower lobe was freed up and released. The reinflation was carried out as in Part I. Sections of the atelectatic accessory lobe were taken for microscopic study. Any lobes showing evidence of gross infection were discarded from this series.

In one experiment the lungs were removed from a stillborn human fetus of seven months within four hours after birth. These lungs were found grossly and microscopically normal except for complete atelectasis. Reinflation pressures were determined in the negative pressure respirator in the manner used for the above experiments.

Results—Experiments varying in duration of atelectasis from two to twenty-six days were carried out in 13 cats. The results are tabulated in Table III.

In chronic atelectasis up to twenty-six days' duration (beyond which

TABLE III

EXPERIMENT	DURATION IN DAYS	TYYE OF ANESTHESIA	TYYE OF PRESSURE	PRESSURE	
				INITIAL	FINAL
1	2	nembutal	negative	−16	−20
5	7	nembutal	negative	−18	−20
37	7	ether	negative	−14	−20
14	10	nembutal	negative	−16	−24
36	10	ether	negative	−14	−22
6	11	nembutal	negative	−17	−20
7	14	nembutal	negative	−16	−22
29	15	nembutal	positive	+18	+20
34	18	ether	positive	+16	+22
13	18	nembutal	negative	−14	−22
31	23	ether	positive	+19	+21
32	23	ether	positive	+18	+22
17	26	nembutal	negative	−20	−24
--	--	(fetal atelectasis)	negative	−12	−20
			Mean:	±16	±21

we have not carried the experiments in an uninfected state) the pressures necessary for reinflation were not significantly different from those necessary for reinflation in acute atelectasis. No significant differences were observed in the results following the use of ether or nembutal anesthesia.

DISCUSSION

The existence of a threshold pressure which must be reached before the reinflation of atelectatic lung can proceed is of some clinical significance in the prophylaxis and treatment of atelectasis, and in the postoperative pulmonary complications associated with atelectasis. The three species of animals studied show a close relationship in the threshold pressures. We have not been successful in obtaining at autopsy a specimen of acquired lobar atelectasis in adult human lungs, since uncomplicated lobar atelectasis is a rare cause of death in adults. However, it will be observed that in the atelectatic lungs from a seven month stillborn fetus the effective reinflation pressures were −12 initial and −20 final. While these absolute values obtained experimentally in animals cannot be assumed literally for atelectasis in the intact adult human chest, the conclusion is clear that any method which aims at the reinflation of atelectatic foci must be controlled to give an effective pressure differential.

An effective increase in the depth of the negative intrapleural pressures must occur ordinarily with hyperventilation induced by chemical means. With debilitated individuals, however, and with the inevitable localized restrictions in respiratory excursion due to such factors as abdominal distention, pain, and the position of the patient, it is easy to see how the effective differential pressure might not be created in all parts of the lung field. Positive pressure inhalations with air-carbon-dioxide mixtures by means of a tight mask and a graduated expiratory valve should overcome this objection, as would the simple blow-bottle in patients whose physical and mental condition would permit its employment.

In another communication[11] we have demonstrated the superiority of slight degrees of expiratory resistance over simple hyperpnea for increasing the passage of air into an obstructed lung lobule by collateral respiratory channels. Moreover, studies on the subtidal lung volume in anesthetized dogs show a considerable increase in the lung volume with small degrees of expiratory resistance.[12]

Upon these experimental grounds we propose the use of positive pressure breathing, with air-carbon-dioxide mixtures, in the routine prophylaxis and treatment of atelectasis.

CONCLUSIONS

1. A threshold differential pressure exists for the reinflation of atelectasis, experimentally produced in animals.

2. In four species studied, cat, dog, rabbit, and human (fetal), this pressure was from 12 to 16 cm. water (average 14).

3. If this pressure is not reached or exceeded, reinflation does not occur.

4. The effect of chronicity in the atelectasis (up to twenty-six days) was not to raise the threshold, or to raise it only to an insignificant degree (2 cm. average).

5. The type of operative anesthesia (ether or nembutal) had no demonstrable effect on the threshold pressure.

6. Reinflation, as indicated by the effective pressures, proceeded as readily by the collateral as by the direct bronchial route.

7. The use of a positive pressure control in prophylactic or therapeutic inhalations in combating atelectasis is proposed.

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THE EFFECT OF EXERCISE ON THE ACID-BASE BALANCE AND OXYGEN OF THE BLOOD FOLLOWING ATELECTASIS AND PNEUMECTOMY*†

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THE ability of the organism to adjust itself to the functional or anatomic removal of a large fraction of its pulmonary tissue is well established. As a matter of fact, an animal at rest appears entirely normal when 50 per cent of its lung tissue has been removed. It seemed of interest to determine to what extent such animals could adjust themselves to conditions in which the respiratory apparatus was put under severe strain. It is believed that such information would be of importance in evaluating the physiologic effect of reduction in the amount of pulmonary tissue.

In order to determine this the following experiments were performed. Partial lung collapse, corresponding to the functional removal of approximately 50 per cent of the lung tissue, was produced in 11 dogs, and partial pneumectomy was performed on five dogs. At intervals varying from one day to fourteen months after the operation, the response of the animals to exercise was determined.

Heuer and Andrus[1] studied the effect of pneumectomy on dogs and found that from four to six weeks after the removal of one lung the remaining lung practically filled the entire pulmonary cavity. They also made frequent observations of the alveolar oxygen and carbon dioxide, and of the carbon dioxide and oxygen content and capacity of the venous blood after the pneumectomy. They found that the alveolar carbon dioxide was higher than normal, and the alveolar oxygen was lower than normal, about ten or eleven days after the pneumectomy. Following this the values were approximately normal. An increase in oxygen capacity was also observed about two weeks after the operation. Since most of our animals had been operated upon several months before they were exercised, the compensatory changes observed by Heuer and Andrus had already occurred before the experiments we are reporting were performed upon them.

Since reduction in the efficiency of the respiratory apparatus when placed under strain would be reflected in the amounts of oxygen and carbon dioxide carried by the blood, determinations were made of the

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*Received for publication, September 8, 1933.
†This work was in part supported by a grant from the Douglas Smith Foundation.

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degree of saturation of the arterial blood with oxygen and the direction and extent of the displacement of the acid-base balance of the blood from normal.

Three types of exercise were used: (1) running on a motor-driven treadmill; (2) swimming in a tank at 30° C.; and (3) swimming at 40° C.

A comparable series of experiments was performed on normal dogs.

EXPERIMENTAL

Details of Operative Technic

Massive atelectasis of one or more of the pulmonary lobes was brought about by completely occluding the primary bronchus to that lobe.[2] This was accomplished by cauterizing the entire circumference of the bronchus through a bronchoscope with a 35 per cent solution of silver nitrate. About two weeks subsequent to the cauterization, complete stenosis of the bronchus resulted and was accompanied by massive atelectasis of the obstructed pulmonary tissue. The entire thorax was filled by hyperinflation of the remaining lobes.

Lobectomy or pneumectomy was performed subsequent to collapse of the part to be excised.[3] The lung tissue was removed without difficulty through a thoracotomy opening in the fourth or fifth rib-bed, subsequent to a subperiosteal resection of a small portion of that rib. The pulmonary vessels and bronchus were doubly ligated and divided between ligatures, the bronchus being ligated distal to the region of stenosis to control bronchial bleeding.

An estimate of the reduction in active pulmonary tissue was based on data obtained from weighing individual pulmonary lobes in a series of 15 dogs, and calculating the relationship of each lobe to the entire lung.

Plan of Experiments

In all three types of exercise employed, an attempt was made to keep the procedure essentially the same, although in practice this was not always possible. A sample of blood was drawn from a femoral artery before the experiment. After ten minutes of exercise, a second sample of blood was drawn; after thirty minutes, a third sample; and a fourth sample after approximately ten minutes' rest following the exercise.

The blood was drawn without exposure to air and divided into two portions. One was transferred to a tube under oil with potassium oxalate for the determination of its oxygen content and capacity. The other was transferred to a centrifuge tube under oil and centrifuged. The carbon dioxide content and P_H of the serum were then determined.

The oxygen and carbon dioxide determinations were made on a Van Slyke and Neill manometric blood gas apparatus. The P_H was determined by the colorimetric method of Hastings and Sendroy. The rectal temperature of the animals was recorded in most cases.

TABLE I

TREADMILL EXERCISE

DOG NO.	P_H	SERUM		CO_2 TENSION	OXYGEN CONTENT	OXYGEN CAPACITY	PERCENTAGE SATURATION	BODY TEMP.	REMARKS	ESTIMATED PERCENTAGE NORMAL LUNG
		CO_2	HCO_3							
		MM/L	MM/L	MM. HG	C.C./100	C.C./100		°C.		
					NORMAL					
2	7.30	17.7	16.6	34.8	17.5	19.3	91	39.0	At rest	100
	7.28	18.1	17.0	37.5	15.3	17.9	86	39.9	15 min. exercise	
	7.27	17.5	16.4	36.8	15.0	18.8	80	—	7 min. rest	
	7.27	17.9	16.8	37.6	18.0	18.3	98	—	19 min. rest	
					PARTIAL LUNG COLLAPSE					
8	7.38	22.4	21.3	37.0	22.8	23.7	96	39.0	At rest—9 months after operation	43
	7.38	22.3	21.2	36.7	22.6	24.5	92	40.2	13 min. exercise	
	7.38	21.6	20.5	35.7	22.8	25.5	90	—	9 min. rest	
					PARTIAL PNEUMECTOMY					
16	7.33	22.0	20.8	40.8	20.7	23.3	89	39.2	At rest—10 months after operation	40
	7.29	19.6	18.4	39.6	18.8	22.8	82	40.1	6 min. exercise	
	7.31	20.3	19.1	39.2	19.7	22.3	88	39.0	9 min. rest	

Results

The results of the experiments are given in Tables I to III and in Fig. 1.

Treadmill Exercise.—It was found that running on a horizontal treadmill developed no changes in the blood of the dogs with reduced pulmonary tissue which were greater than those encountered in normal dogs. Five such experiments were performed, of which the results of three are given in Table I to illustrate the changes observed. There seems to have been a decrease in the saturation of the arterial blood with oxygen after the exercise in all cases, but this was no more marked in the operated than in the normal animals. The changes in the acid-base balance were too slight to be of significance.

Swimming at 30°.—The effect of swimming in a tank of water maintained at 30° C. was then studied. The results of these experiments are given in Table II. No significant change in the saturation of the blood with oxygen or in the acid-base balance was observed in the normal dog. This is in accord with the observations of Hastings and Steinhaus, who found that no material change in the acid-base balance occurred when dogs swam in water at 30°.[4]

In animals with reduced pulmonary tissue some changes in acid-base balance were observed, however. This is most clearly shown in the column marked ΔA^-. This represents the change in fixed acids produced by the exercise and is calculated as follows: It is the difference between the initial serum HCO_3 concentration and the HCO_3 concentration at any other time, calculated to the initial P_H.

$$\Delta A^- = \Delta HCO_3 = (HCO_3)_i - (HCO_3)_{\epsilon i}$$

where $(HCO_3)_i$ = the initial HCO_3 concentration,

and $(HCO_3)_{\epsilon i}$ = the HCO_3 concentration at a subsequent time, calculated to that which it would have been at the initial P_H.

$$(HCO_3)_{\epsilon i} = (HCO_3)_{\epsilon} - (\beta \times \Delta P_H)$$

where $(HCO_3)_{\epsilon}$ = the observed HCO_3 concentration at any given P_H;

β = the buffer value of true serum = 29 for normal blood;

and ΔP_H = the difference between the initial and the experimental P_H.

For example: From the data of Dog 7, initially and after ten minutes of exercise:

$$P_H = 7.33 - 7.31 = 0.02$$
$$(HCO_3)_{\epsilon i} = 15.7 - (29 \times 0.02) = 15.1$$
$$\Delta A^- = 18.0 - 15.1 = 2.9$$

TABLE II

SWIMMING AT 30° C.

DOG NO.	P_H	SERUM CO_2 MM/L	SERUM HCO_3 MM/L	$\triangle A$- MM/L	CO_2 TENSION MM. HG	OXYGEN CONTENT C.C./100	OXYGEN CAPACITY C.C./100	PERCENTAGE SATURATION	BODY TEMP. °C.	REMARKS	ESTIMATED PERCENTAGE NORMAL LUNG
					NORMAL						
5	7.31	19.2	18.1		36.8	15.7	17.2	91	39.8	At rest	100
	7.31	18.7	17.6	+ 0.5	35.8	15.1	16.9	90	40.0	30 min. exercise	
	7.30	19.2	18.1	+ 0.3	37.7	15.0	16.9	89	—	10 min. rest	
					PARTIAL LUNG COLLAPSE						
7	7.33	19.1	18.0		35.2	22.0	22.5	98	39.0	At rest—40 days after operation	57
	7.31	16.7	15.7	+ 2.9	32.1	20.6	22.6	91	—	10 min. exercise	
	7.29	19.1	17.9	+ 1.3	38.4	18.7	22.1	85	41.0	30 min. exercise	
	7.30	17.9	16.8	+ 2.1	34.9	19.4	22.1	88	—	9 min. rest	
8	7.40	21.5	20.5		34.0	—	—	—	38.6	At rest—12 months after operation	43
	7.32	21.4	20.2	+ 2.6	40.5	—	—	—	—	10 min. exercise	
	7.38	21.4	20.3	+ 0.8	35.4	—	—	—	40.4	30 min. exercise	
	7.39	21.1	20.1	+ 0.7	34.2	—	—	—	—	10 min. rest	
					PARTIAL PNEUMECTOMY						
12	7.33	19.0	17.9		35.1	17.1	19.5	88	38.7	At rest—2 months after operation	57
	7.13	11.0	10.1	+13.6	31.0	17.2	19.5	88	—	10 min. exercise	
	7.28	17.2	16.1	+ 3.3	35.2	16.8	18.2	92	39.6	30 min. exercise	
16	7.34	22.1	20.9		39.7	25.2	26.1	96	39.0	At rest—13 months after operation	40
	7.21	21.6	20.1	+ 4.6	51.5	24.1	25.5	95	—	10 min. exercise	
	7.22	22.6	21.0	+ 3.5	53.0	23.4	25.5	92	39.5	30 min. exercise	
	7.31	20.9	19.7	+ 2.1	40.7	23.7	25.9	92	—	10 min. rest	

TABLE III

SWIMMING AT 40° C.

DOG NO.	P_H	SERUM CO_2 MM/L	SERUM HCO_3 MM/L	ΔA- MM/L	CO_2 TENSION MM. HG	OXYGEN CONTENT C.C./100	OXYGEN CAPACITY C.C./100	PERCENTAGE SATURATION	BODY TEMP. °C.	REMARKS	ESTIMATED PERCENTAGE NORMAL LUNG
					NORMAL						
4	7.38	18.6	17.7		30.8	—	—	—	39.0	At rest	100
	7.32	13.7	12.9	+ 6.5	25.7	—	—	—	—	10 min. exercise	
	7.42	11.8	11.3	+ 5.2	11.4	—	—	—	—	30 min. exercise	
	7.36	13.4	12.7	+ 5.6	22.8	—	—	—	—	10 min. rest	
7	7.25	17.8	16.6		38.7	—	—	—	39.7	At rest	100
	7.23	12.5	11.6	+ 4.7	28.3	—	—	—	42.6	30 min. exercise	
	7.33	10.7	10.1	+ 4.2	19.4	—	—	—	—	7 min. rest	
	7.24	10.1	9.4	+ 7.5	22.2	—	—	—	—	13 min. rest	
7	7.30	19.2	18.1		37.8	16.3	18.2	90	39.6	At rest	100
	7.30	15.9	15.0	+ 3.1	31.3	—	—	—	—	10 min. exercise	
	7.30	13.6	12.8	+ 5.3	26.6	15.7	20.0	80	42.1	30 min. exercise	
	7.30	14.8	13.9	+ 4.2	29.0	—	—	—	—	15 min. rest	
					PARTIAL LUNG COLLAPSE						
9	7.38	21.7	20.6		35.9	—	—	—	39.7	At rest—14 months after operation	57
	6.95	4.2	3.7	+29.4	15.9	—	—	—	—	9 min. exercise	
	6.95	3.9	3.4	+29.7	14.9	—	—	—	42.8	14 min. exercise	
	6.95	3.6	3.1	+30.0	38.3	—	—	—	—	8 min. rest	
10	7.41	24.6	23.5		38.3	—	—	—	38.9	At rest—11 months after operation	57
	7.41	9.6	9.1	+14.4	16.8	—	—	—	—	10 min. exercise	
	7.40	12.3	11.7	+12.1	19.2	—	—	—	43.8	15 min. exercise	
	7.41	14.0	13.3	+10.2	21.6	—	—	—	—	10 min. rest	
12	7.39	22.9	21.8		37.0	—	—	—	38.1	At rest—6 months after operation	57
	7.28	10.0	9.4	+15.6	20.2	20.2	24.3	83	—	10 min. exercise	
	7.20	11.1	10.3	+17.0	26.7	18.8	21.9	86	42.4	17 min. exercise	
	7.30	9.2	8.7	+14.7	17.7	19.7	22.9	86	—	9 min. rest	

TABLE III—Cont'd

DOG NO.	P_H	SERUM CO_2 MM/L	SERUM HCO_3 MM/L	ΔA- MM/L	CO_2 TENSION MM. HG	OXYGEN CONTENT C.C./100	OXYGEN CAPACITY C.C./100	PERCENTAGE SATURATION	BODY TEMP. °C.	REMARKS	ESTIMATED PERCENTAGE NORMAL LUNG
13	7.42	17.4	16.6		26.4	—	—	—	40.0	At rest—8 months after operation	57
	7.19	15.3	14.2	+ 9.1	37.5	—	—	—	—	10 min. exercise	
	6.90	19.4	16.8	+14.9	88.3	—	—	—	42.8	20 min. exercise—Died	
14	7.33	18.3	17.3		33.9	—	—	—	39.3	At rest—11 months after operation	57
	7.34	9.2	8.7	+ 8.3	16.7	—	—	—	—	10 min. exercise	
	7.12	7.2	6.6	+16.8	20.8	—	—	—	42.3	30 min. exercise	
	7.23	6.8	6.3	+13.9	15.6	—	—	—	—	11 min. rest	
15	7.30	22.0	20.7		43.3	24.4	25.8	94	39.3	At rest—8 months after operation	57
	7.21	10.4	9.7	+12.6	23.8	23.9	26.4	91	—	10 min. exercise	
	7.30	14.4	13.5	+ 7.2	28.2	22.9	25.8	88	42.3	30 min. exercise	
	7.30	15.6	14.7	+ 6.0	30.7	—	—	—	—	11 min. rest	
					PARTIAL PNEUMECTOMY						
12	7.40	22.0	21.0		33.9	—	—	—	39.6	At rest—6 months after operation	
	7.44	17.1	16.4	+ 3.4	24.7	—	—	—	42.1	30 min. exercise	
	7.40	18.6	17.7	+ 3.3	29.3	—	—	—	—	10 min. rest	
15	7.37	16.4	15.6		27.8	—	—	—	39.5	At rest—6 months after operation	57
	7.31	9.2	8.7	+ 8.6	17.8	—	—	—	—	10 min. exercise	
	7.38	12.5	11.9	+ 3.4	20.7	—	—	—	43.6	30 min. exercise	
	7.38	14.2	13.5	+ 1.8	23.5	—	—	—	—	7 min. rest	
7	7.40	22.6	21.5		35.6	—	—	—	39.6	At rest—12 days after operation	57
	7.45	15.5	14.8	+ 5.2	21.8	—	—	—	—	10 min. exercise	
	7.42	16.1	15.4	+ 5.5	24.4	—	—	—	—	30 min. exercise	
	7.42	12.9	12.3	+ 8.6	19.4	—	—	—	—	10 min. rest	
17	7.35	22.5	21.3		39.5	24.4	26.0	94	38.8	At rest—10 months after operation	24
	7.04	8.4	7.5	+22.8	28.0	21.5	26.4	82	41.9	19 min. exercise	
	7.19	13.8	12.8	+13.1	32.8	21.5	25.7	81	—	13 min. rest	

In the animals with partial pulmonary collapse, the fixed acids of the blood had increased 2.6 and 2.9 millimols per liter after ten minutes' swimming. After swimming for twenty minutes longer, the fixed acids were only slightly increased. This has been previously observed and is associated with the adjustment of the circulatory and respiratory mechanisms to the demand of the muscles for oxygen. The changes in the CO_2 tension were not of sufficient magnitude to be of significance.

In the two animals with partial pneumectomy there was an even greater increase in fixed acid in the ten-minute samples. In addition, in one of the dogs, Dog 16, there was a marked increase in the CO_2 tension of the blood in the ten- and thirty-minute samples, indicating a failure of the respiratory mechanism to adequately remove CO_2.

The change in percentage saturation of the arterial blood with oxygen was slight and inconsistent in direction. On the basis of these data it is not possible, therefore, to attribute the greater change in fixed acids in the experiments on the dogs with reduced respiratory tissue to inadequate oxygenation of the blood.

Swimming at 40°.—Since treadmill exercise and swimming at 30° failed to elicit differences of significance between the normal dogs and those with reduced pulmonary tissue, a series of experiments was made in which the exercise was of a more severe character, namely, swimming in water at 40° C. This had been found by Hastings and Steinhaus to be accompanied by both fixed acid excess and CO_2 deficit in normal dogs. Three experiments on normal dogs, eight experiments on dogs with partial lung collapse, and four experiments on animals with partial pneumectomy were therefore performed. The data of these experiments are given in Table III. Since two of the dogs with partial lung collapse swam only ten minutes, after which one died, the protocols of their experiments are omitted.

The acid-base balance changes were marked in all cases. These changes were (1) a marked increase in fixed acids, and (2) a decrease in CO_2 tension. Furthermore, the fixed acid increases in the animals with partial lung collapse were much greater than in the normal animals. In the animals with partial pneumectomy, the fixed acid increase was very marked in one case (Dog 17), but in three other experiments the increase in fixed acids was no greater than it was in the normals. Unfortunately oxygen determinations were made in too few experiments of this series to permit the drawing of conclusions regarding the percentage saturation of the arterial blood.

A marked rise in body temperature accompanied the swimming in all cases.

DISCUSSION

The acid-base changes in the experiments on swimming at 40° can be most clearly illustrated by reference to the acid-base charts (Fig. 1).

In Chart 1, the approximate paths taken by the acid-base balance of the blood for the four conditions, acid excess, alkali excess, CO_2 excess and CO_2 deficit are indicated. The acid-base path of the dog swimming at 40° of Hastings and Steinhaus is also shown for reference. The direction indicates both fixed acid excess and CO_2 deficit. In Chart 2, the results of our three experiments on normal dogs are plotted. It is seen that here, too, the path taken is in the direction of fixed acid excess and CO_2 deficit. The magnitude of the displacement from normal is not so great as in the Hastings-Steinhaus experiment because the exercise was not of such long duration.

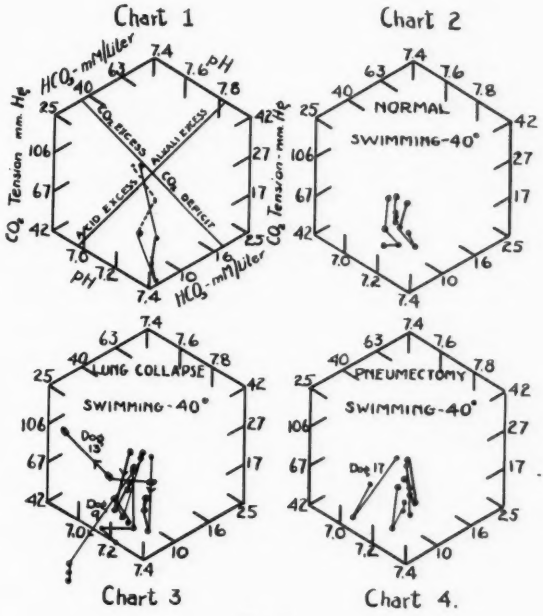

Fig. 1.

In Chart 3 are plotted the experiments on dogs with partial lung collapse. In all except Dogs 9 and 13, the direction taken is the same as in the case of the normal animals, but the extent of the displacement is greater, although the severity of the exercise was the same. This indicates (1) that the rate of entrance of fixed acids into the blood stream was greater in these animals than in the normals and (2) that the respiratory mechanism was adequate to maintain the elimination of CO_2 at a normal level. In one animal (Dog 9) the production of fixed acids was excessive, and the overbreathing was not so great as would have been expected on the basis of the experiments on the normal animals. This is shown by the fact that the path, instead of being in the direction

of 6 o'clock on the chart, was in the direction of 7 o'clock. In Dog 13, respiratory difficulties were present from the start, as indicated by the fact that the acid-base path took the direction of fixed acid excess and CO_2 excess after ten minutes of exercise and the direction of CO_2 excess during the subsequent ten minutes of swimming. This animal died soon after.

In Chart 4 are plotted the experiments on dogs with partial pneumectomy. In three of these, the direction and magnitude of the displacement are similar to those in the experiments on normal dogs. In the fourth, Dog 17, the direction is that of fixed acid excess without CO_2 deficit. This animal had lost three-fourths of its lung tissue, and therefore the efficiency of the removal of CO_2 by overventilation had been materially reduced.

CONCLUSIONS

One may draw the following conclusions from these experiments:

1. That reduction in active pulmonary tissue by 50 per cent does not embarrass the organism when engaged in moderate exercise, such as running on a level.

2. That more strenuous exercise such as swimming results in more fixed acid production than normal. This probably is to be interpreted as indicating that the tissues are receiving a less than normal supply of oxygen and incomplete oxidation results. A decreased percentage saturation of the blood with oxygen in some instances would tend to favor such an explanation, although these results are too incomplete to provide conclusive proof.

3. That the elimination of CO_2 appears to proceed with normal efficiency unless as much as 75 per cent of the active pulmonary tissue is removed. Even then severe exercise seems to be well tolerated.

4. That when the active pulmonary tissue was reduced by equal amounts, fixed acid increase was more marked in animals with partial collapse than in those with partial pneumectomy, suggesting that a significant fraction of the blood flows through the atelectatic tissue.

SUMMARY

1. The acid-base balance and percentage saturation of the arterial blood of dogs whose active pulmonary tissue had been reduced have been determined before and after exercise of varying amounts and varying degrees of severity.

2. There was no difference between the response of the operated and unoperated dogs to mild exercise.

3. Swimming caused a greater increase in fixed acids in the dogs with reduced pulmonary tissue than in the normal dogs.

4. Dogs with 50 per cent of their active pulmonary tissue removed

apparently have no difficulty in eliminating carbon dioxide even under stress, and but slight difficulty in delivering an adequate supply of oxygen to their tissues.

5. Partial lung collapse leads to acid-base changes on exercise of much greater magnitude than does comparable partial pneumectomy.

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SUPERIOR VENA CAVAL OBSTRUCTION WITH A CONSIDERATION OF THE POSSIBLE RELIEF OF SYMPTOMS BY MEDIASTINAL DECOMPRESSION*†

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PATIENTS with obstruction of the superior vena cava are very likely to die as a result of that obstruction unless a satisfactory collateral circulation becomes established. But even if they do not die from the obstruction, the effects of it, such as cyanosis, dyspnea, edema of the face and upper trunk, and increased venous pressure, are most distressing. It is not sufficiently realized that sometimes marked relief from the distressing symptoms can be obtained by the comparatively simple procedure of a decompression operation on the mediastinum. The type of case in which a beneficial result is most likely to occur is that in which the obstruction is caused by the outside pressure of a tumor. On the other hand, an unsatisfactory result will be obtained if the tumor is one which encircles the vein and therefore obstructs by constriction, or if the lumen is blocked either by the actual presence of tumor tissue within it or by a thrombus. We have, therefore, undertaken to ascertain just how often the condition is amenable to treatment. It has also been deemed advisable to scrutinize the recorded cases to determine whether or not the evidence thus far presented permits one to state that complete occlusion of the superior vena cava is compatible with life. More exact information upon this point is afforded by the recent experimental work of Herbert Carlson.

REVIEW OF THE LITERATURE

Corvisart, in 1806, was the first to report upon narrowing of the superior vena cava of a high degree. The first case of complete obliteration was reported by Marjolin, in 1819. These were followed by a series of inaugural dissertations by Deckart (1823), Weissbrod (1831), Pöschel (1903), Fischer (1904), Pawel (1910), Rauth (1911), and by Steib (1920).

Oulmont, in 1856, reported a small series of cases, while Fischer, in 1904, reported upon 252 collected cases; 226 of these patients had come to necropsy and complete obliteration is said to have occurred in 104. Seven years later, Rauth was able to collect 277 cases, and reported 4 personal experiences. According to Dana 24 additional cases were

*Received for publication April 1, 1933.

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reported between the years 1911 and 1920, and since 1922 we have been able to collect 8 others. These were reported by Ruitinga, Berblinger, Brannan, Foot, Jonkhoff and Geerling, Strauss Brown, and van der Hoeven. Up to the present time, therefore, the literature contains reference to 309 cases. All of these, however, are not examples of complete occlusion. Moreover, the signs of partial occlusion are so common in cases of mediastinal tumors of all kinds that probably many cases have not been reported. To this number of 309 cases collected from the literature, we wish to add one case of proved complete occlusion and another in which there was a very high grade obstruction (possibly complete) which was relieved by decompression.

Of the 220 cases reported by Fischer 71.31 per cent occurred in males and 28.6 per cent in females.

The age periods at which the condition was observed in the group of patients referred to by Fischer were as follows:

TABLE I

AGE PERIOD	NO. CASES
0-10 years	3
11-20 years	12
21-30 years	33
31-40 years	38
41-50 years	71
51-60 years	33
61-70 years	27
71-80 years	3

TABLE II

GROUP	ETIOLOGIC FACTOR	PER CENT
I	Propagating thrombi from periphery	4
II	Localized phlebitis with thrombus formation	1.5
III	Tuberculous phlebitis	1.5
IV	Actinomycosis	0.5
V	Traction or compression by scar tissue	6
VI	Obliteration of the pericardium, with consequent narrowing of the superior vena cava by cardiac and respiratory movements	0.5
VII	Thoracic tumors	
	A. Primary thoracic tumors	
	1. Benign	4
	2. Malignant	37
	B. Extrathoracic malignant tumors	
	1. Metastatic to thoracic organs	4.25
	2. Tumors which break into the tributaries of superior cava and grow down into it	4.25
	3. Metastasis into vein wall itself	0.5
	C. Aneurysms	36

It can be noted from Table I that 32 per cent of the cases reported by Fischer occurred in patients between the ages of forty-one and fifty years. This coincides fairly well with the incidence of aneurysm

for this age period. It should also be remembered that a relatively large number of malignant tumors are observed during this decade.

The etiologic factors in the cases collected by Fischer have been classified as shown in Table II. Reference is also made to the relative frequency of each factor.

Brown, in a brief summary of the findings in some 300 collected cases, estimated that aneurysm of the aorta and mediastinal tumors were equally responsible for about 55 per cent of the cases. Carcinoma of the lung, bronchi, and adjacent organs caused obstruction in about 25 per cent of the cases. The remaining 20 per cent of the cases were attributable to the various less frequent causes (Groups I-VI), as described by Fischer.

Morbid Anatomy.—The pathologic processes causing superior vena caval obstruction listed in the first six groups described by Fischer, are, on the whole, self-explanatory and require no discussion. A few remarks may well be made concerning the last group of etiologic factors which includes primary benign and malignant tumors, metastatic tumors, and aneurysms.

The consequenses of variations in increased intramediastinal pressure have been referred to by Ballon and Francis. It is perhaps well to reiterate, however, that, irrespective of the mechanism, any serious encroachment upon the mediastinal contents must of necessity produce changes which are either local or general. Such changes may vary and are dependent not only upon the normal arrangement of the anatomic structures in the region concerned, but also upon their resistance to stresses and strains. Thus, many of the consequences of variations in mediastinal pressure may be the result of compression of the larger blood vessels. It is frequently much easier for compression of the large blood vessels to occur than of the trachea or bronchi. Cyanosis and dyspnea too are frequently expressions of compression of the former.

Benign tumors may compress the superior vena cava because of their size and because they may result in increased intramediastinal pressure; for it is certain that in a closed thorax one structure cannot expand to any great extent save at the expense of another.

Obstruction of the superior vena cava by primary malignant or metastatic tumors may result from external compression, from obliteration by the growth of the tumor in the lumen of the vein or from the simultaneous action of both. When due to carcinoma Fischer found that practically one half the specimens revealed actual invasion of the wall of the vessel and of its lumen. In the remainder, the obstruction was due to compression from without, with or without thrombosis. The obstruction from without may be due either to pressure from one direction or to a constriction caused by an encircling growth.

In one of the cases referred to in the present communication, the obstruction of the superior vena cava was due to metastases from a primary teratoma of the testicle. Up to the present time no similar case is to be found in the literature. Because it was demonstrated satisfactorily at postmortem that a complete occlusion was present and that a collateral circulation had been established, this case is reported in some detail.

CASE 1.—H. C. N., a white male, aged forty-seven years, a car finisher by occupation, entered Barnes Hospital on November 24, 1931. His complaints were extreme dyspnea (orthopnea), swelling of the right arm and face, and cyanosis. About two months prior to his admission he had noted edema of the left arm which had lasted but two days. One month later he developed a productive cough, raising small quantities of bloody mucoid sputum. At this time too his face and neck felt "thick." During the week of November 17, 1931, while firing furnaces, he became very weak and dizzy, requiring medical aid. Immediately after this, his face and right arm began to swell, and he soon became orthopneic and cyanotic.

Family history: negative. *Past history:* irrelevant. *Marital history:* married twice; first wife died of pulmonary tuberculosis.

Physical Examination.—Patient deeply cyanotic from waist up. There was a band of ecchymosis two inches wide which encircled the entire chest at the level of the ensiform cartilage. Face, neck, arms, and thorax markedly "swollen"; thorax anteriorly exhibited many venules at the level of the sixth and eighth ribs. Fundi showed tremendous distention of the veins, but no swelling of the discs.

Venous pressure in both upper extremities registered 72 cm. of water. (The normal pressure according to the method used does not exceed 8 to 10 cm.) Blood pressure in the left arm 120/65, in the right 120/?; in both legs 155/110. Physical signs referable to the chest were indefinite save for increased mediastinal dullness to percussion in the second interspace, left. The heart was apparently normal, but the electrocardiogram revealed changes suggestive of myocardial damage.

The abdomen was normal save for a small hard, discrete mass in the subcutaneous tissue, just above and to the right of the umbilicus. The *right testicle* which was not so large as the left one, contained a small hard, tender nodule.

There were no abnormal peripheral pulsations. The edema present was not of the pitting type. The reflexes were all normal.

The roentgen-ray film of the chest taken on November 20, 1931, showed nodules throughout both lungs and suggested pleurisy at the right base. (Fig. 1.)

Diagnosis.—Tumor of the right testicle (probably a teratoma) with metastases to both lungs, to the right pleural cavity, and to the mediastinum; questionable metastases to the abdominal wall.

Treatment.—Appreciating that teratomas of the testicles are radiosensitive, the patient was subjected to deep x-ray therapy. It was hoped that this treatment might relieve him of those symptoms which were obviously due to obstruction or compression of the superior vena cava. Unfortunately, this did not occur. In a very short time he developed fluid in both pleural cavities and repeated aspirations became necessary. Rather large quantities of fluid were removed but the aspirations relieved him but little. The patient became markedly cyanotic and required oxygen therapy. He developed signs of pneumonia in the right upper lobe, and died on February 12, 1932.

Necropsy Findings.—The clinical diagnosis was confirmed. There was a tumor in the right testicle. It proved to be a teratoma. The nodules in the lung were metastases. They contained most of the tissue elements found in the primary lesion; viz., adenocarcinoma, cartilage, and bone. There was a small amount of fluid

in each pleural cavity. Liver and spleen were found to be enlarged but were free from metastases.

Mediastinum.—The posterior mediastinal space was found to be occupied by a nodular mass about 8 by 4 by 4 cm. in size. The thoracic aorta was firmly bound down to this mass which also pressed upon the esophagus and at one point occluded it to such an extent as to admit only the index finger.

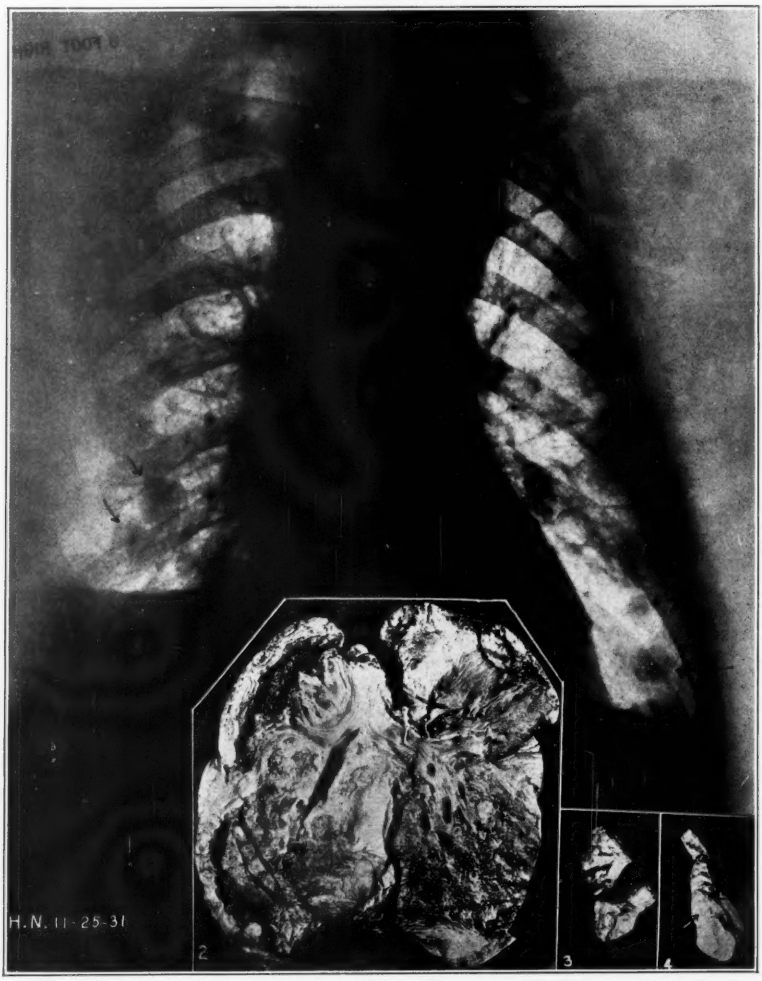

Fig. 1. (Case 1) Superior vena cava obstruction due to metastatic teratoma of testicle. Note the nodules throughout both lungs and the evidence of pleurisy at the right base. The mediastinal shadow in the upper half of the chest is rather wide. *2*, Cross-section of right lung. *3*, represents the thrombosed superior vena cava in cross-section and *4* the nodule in the right testicle.

The *superior vena cava* was found to be patent above the auricle as far as the azygos vein, but it was somewhat narrowed. About 4 cm. above the auricle it was

found to be filled with a greyish white extremely friable cellular tissue. This cellular mass extended to the confluence of the innominate veins. The left innominate vein was found filled with a fine yellowish white material which was adherent to the walls of the veins. The right innominate vein was occluded by a similar thrombus. The left innominate and subclavian veins were attached by their upper surfaces to tumor nodules in the mediastinum. There were many tumor nodules along the vertebral column. They extended down through the diaphragm. On cut section they presented the same appearance as the tumor mass in the mediastinum. The mass in the right abdominal wall was composed of lipoid tissue.

Discussion.—To us the most significant finding was the fact that upon microscopic examination the superior vena cava was found to be completely occluded by thrombus and tumor tissue. Apparently no blood passed through the obstruction.

A search through the literature reveals the fact that necropsy findings of so-called complete obliteration of the superior vena cava are in practically every case based solely upon gross inspection of the specimen and the inability to pass a probe past the point of obstruction. In fact, in but few instances are the microscopic findings in the obstructed vein recorded; and they did not always permit one to exclude the possibility of recanalization of the vein. Most descriptions are limited to the remark "the lumen is completely occluded by an organized thrombus." Baumgarten, in his study of the organization of the thrombus, included recanalization in the term "organization." Osler, in referring to the necrospy findings in the case reported by Wilkins, says, "The superior vena cava was found to be obliterated. . . . On section of the vein it was found converted into a firm fibrous cord, the ultimate result of a thrombus in the vessel." On microscopic examination of the vein, however, he described a definite recanalization and vascularization which he compares to the process of organization of a thrombus described by Baumgarten. There is clearly confusion in terminology. With no desire to be captious, the point must be made that a vein which is organized and recanalized cannot always be considered as completely occluded. Just how effective the recanalization of a thrombus is in draining an area previously taken care of by a vessel of the relatively enormous dimensions of the superior vena cava, is difficult to say. Undoubtedly the marked dilatation of the vessels in the collateral circulation plays the major rôle in maintaining the economy of the organism. Probably the true significance of recanalization is dependent not only upon the etiologic factor at play but also upon how gradually the occlusion of the superior vena cava takes place; in other words, whether or not sufficient time has elapsed to permit of the development of an adequate collateral circulation.

SIGNS AND SYMPTOMS OF SUPERIOR VENA CAVAL OBSTRUCTION

Superior vena caval obstruction can readily be diagnosed. The obstruction hinders the return flow of blood; this in turn results in tre-

mendous distention of veins, because of greatly increased venous pressure. There then occurs a transudation of plasma, and edema develops. Marked dyspnea and cyanosis are always present at this stage. According to some it is due primarily to a tissue anoxemia, concomitant with the profound reduction in vascular efficiency. Rauth attributes the cyanosis largely to the translucency of the tremendously overdistended smallest subcutaneous vessels. Osler suggested that in the establishment of a collateral circulation, anastomosis between the bronchial and pulmonary vessels probably takes place, resulting in dilatation of these vessels, with thickening of their walls. Conditions less favorable to the interchange of gases in the pulmonary capillaries therefore result. This, he thought, explained the dyspnea which is observed in patients suffering from superior vena caval obstruction. We have been unable to verify this fact in the case already described. The dyspnea can more easily be explained by the chemical changes which occur in the blood consequent to stasis.

Objectively then, a marked cyanosis and swelling of the upper part of the body, including the head, neck, trunk, and upper extremities may be noted. There is in many cases a sharply delimiting line recognizable. The lower extremities are usually not affected unless there is associated cardiac insufficiency or if there be distention of the inferior vena cava (because of the additional burden imposed upon it by the collateral circulation) with some obstruction to its tributaries. In no case is the edema in the lower extremities so marked as it is in the upper part of the body. The degree of these signs and symptoms is dependent upon the formation of a collateral circulation.

The most striking objective signs of superior vena caval obstruction are cyanosis and lividity of the face, suffusion of the conjunctivae, prominent and staring eyes, edema of the neck, arms, thorax, and upper part of the abdomen, edema and cyanosis of the mucous membrane of the tongue, mouth, pharynx, and larynx. Rauth states that edema of the glottis may be the cause of sudden death in some cases. Venous pressure also leads to hydropsic transudation of serum into the serous cavity, causing hydrothorax which is especially marked when the azygos vein is included in the obstruction. It may also lead to hydropericardium. The former increases the dyspnea, the latter leads to early cardiac decompensation. The venous stasis may also cause cerebral symptoms because of edema in the meninges and brain substance or because of distention of the ventricles. Lilienthal gives as subjective symptoms headache, vertigo, deafness, epistaxis, tinnitus, visceral disturbance and somnolence, or temporary loss of consciousness. Chiray and Semelaique report the occurrence of Jacksonian epilepsy and Waterfield of Biot's respiration. Many cases of incomplete obstruction, however, do not present the more striking objective signs. In some the most conspicuous evidence of the condition is the dilated

veins on the anterior wall of the chest, a feature which is discussed in the following paragraph.

COLLATERAL CIRCULATION

Characteristic is the turgescence of the obstructed veins and the prominence and course of the veins in the collateral circulation. The subcutaneous veins on the anterior wall of the trunk form a wide meshed network which establishes connections between the veins of the neck, axilla, and the thigh, and by means of branches going deeply also through anastomoses with the intercostals, internal mammary, deep epigastric and the portal veins (through the paraumbilical veins) and the bladder (Burrow's vein) veins, (Skillern). Fischer (Table III) has worked out the external collateral circulation in superior vena caval obstruction.

TABLE III

EXTERNAL COLLATERAL CIRCULATION IN SUPERIOR VENA CAVAL OBSTRUCTION (FISCHER)

1. Jugular (subcutaneous cervical veins)—external mammary (subcutaneous veins of thorax)—superficial epigastric—femoral—vena cava inferior.	
2. Jugular—external mammary—superficial medial xiphoid—superficial epigastric—femoral—vena cava inferior.	
3. Jugular—external mammary (medial xiphoid)	superficial epigastric superficial circumflex iliac femoral—cava
4. Jugular—external mammary (medial xiphoid)	superficial epigastric superficial circumflex iliac femoral—cava superficial external
5. Axillary—long thoracic—epigastric—superficial epigastric or superficial circumflex iliac—femoral cava.	
6. Anterior jugular—Luschka's anastomosis to internal mammary.	
7. Arcus venosus juguli—anastomosis to internal mammary.	
8. Upper thoracic rami of intercostal veins—descending anastomatic branches along spinal column—deeper thoracic rami—deeper intercostal veins.	

In addition to the external collateral circulation there is also an internal one and this depends more specifically upon the location of the superior vena caval obstruction. Fischer gives three types of superior vena caval obstruction. 1. Only below the azygos orifice—31 of 166 cases. 2. To above the azygos orifice—121 of 166 cases. 3. Only above the azygos orifice—14 of 166 cases.

In Case 1, cited earlier in this article, the obstruction was only above the azygos vein, leaving a patent caval passage from the azygos vein to the heart. This allowed a return of blood through the azygos vein and permitted the establishment of a return flow by way of the internal mammary and by way of the various pathways which lead from the cervical and axillary veins to the intercostals.

The blood from the head, neck, and upper extremities is brought to the intercostals together with that of the thoracic wall itself and is

returned to the cava chiefly through the azygos vein, with the aid of the hemiazygos and hemiazygos accessoria. The internal mammary takes up a part of the blood of the innominate and also a part of the blood of the intercostals and carries it to the inferior vena cava by the pathways indicated above. Another probable route in the collateral circulation, not mentioned in most of the previous papers touching on this matter, is from the skull to the inferior vena cava by way of the sinus sigmoideus and the vertebral venous plexuses (Berblinger).

EXPERIMENTAL FINDINGS OF CARLSON

Carlson found that the dog tolerates obstruction of the superior vena cava above the level of the azygos vein but not obstruction below the level of the azygos, unless carried out in two stages. He experimentally proved that the cyanosis due to superior vena caval obstruction will disappear if an adequate collateral circulation has been established. The paths of collateral circulation have been described by Carlson. It is worthy of note too that pleural effusions developed only when the point of obstruction was above the azygos vein. Carlson's experiments also show that venous pressure is definitely increased when the obstruction is above the azygos vein but that once a collateral circulation develops, it may become normal.

Prognosis.—According to Osler, obliteration of any one of the three great veins of the body may exist for many years and the person remain in good health, provided the collateral circulation is adequate. Gossage, in 1912, reported a case in which the diagnosis was based upon the presence of prominent veins over the abdomen and thorax. There were none of the other signs usually present in the symptoms accompanying proved cases of superior vena caval obstruction. In discussing this report Osler, then at Oxford, stated that with a slow sclerotic process and even with complete fibrous obliteration of the vein there is usually sufficient time for an adequate collateral circulation to develop, and the patient is thus able to live for a long time with but little discomfort. The prognosis is thus dependent chiefly upon two factors: (1) the nature and rapidity of the occlusion and the time allowed for effectual collateral circulation, and (2) the rate of growth or expansion of the pathologic process which is the primary cause of the obstruction.

Treatment.—Very little reference to the treatment of this condition is to be found in the literature. In 1918, Favre, in reporting 4 cases of luetic phlebitis, indicated the efficacy of antiluetic treatment in such cases. In the presence of a positive Wassermann reaction this form of treatment should always be given a trial. Chiray and Semelaique reported symptomatic relief and complete warding off of Jacksonian epileptic seizures in such a case by drawing off 50 to 100 c.c. of blood from the jugular vein. Waterfield relieved Biot's respiration by venipuncture.

Many mediastinal tumors show striking regression in size following deep x-ray therapy. Such treatment should be tried in all cases where tumor is suspected.

Decompression operations have seldom been employed to relieve obstruction to the superior vena cava. This fact is rather surprising, particularly when it is appreciated that almost as many patients with aneurysm of the thoracic aorta die from the consequences of compression of important intrathoracic structures as from rupture of the aneurysm.

The following clinical history demonstrates the dramatic result which may be obtained by a decompression operation. The operation was performed by one of us (E. A. G.) upon a patient suffering from superior vena caval obstruction due to mediastinal Hodgkin's disease.

CASE 2.—The patient, a female, aged twenty-four years, first entered Barnes Hospital on February 20, 1930. She stated that she had an attack of severe pain in the upper right chest six months before admission. From then on she tired easily. About four months previously to admission she developed an unproductive cough and a daily rise in temperature. Her face, neck, and arms gradually became markedly swollen.

Physical Examination.—Respirations were rapid and shallow, and there was marked swelling and cyanosis of both eyelids, the face, neck, and arms. The venous pressure in both arms registered 14 cm. of water. The right pleural cavity was found to contain a considerable quantity of fluid. A large tumor mass was also found to extend from the mediastinum into the right chest. Fluoroscopic and roentgen ray examinations confirmed all the above findings. Heart normal, blood pressure 110/70. Lymph glands in right groin were palpably enlarged. The erythrocytes numbered 4,240,000; the leucocytes 18,900, hemoglobin was 85 per cent, the differential blood count (Schilling) was as follows: juv. 1, stab. 10; segmented cells 75, lymphocytes 9, monocytes 5.

Deep x-ray therapy not only failed to produce any rapid change in the size of the mass or to improve the patient's condition, but instead she became worse. The dyspnea increased so much that a decompression operation was therefore performed as an emergency on May 13, 1930. The patient's condition was so critical that she was unconscious and no anesthetic drug was given. Instead, oxygen inhalations were used, and artificial respiration had to be carried out during the entire operation. The fourth and fifth right costal cartilages were removed from the sternum to the bony portions of the ribs. The mediastinum was then exposed and a hard nodular mass firmly attached to the structures within the chest wall was found. The tumor was freed in several directions from these attachments and lifted forward. Grossly, it appeared malignant; a small portion was removed for microscopic section. The veins on the anterior thoracic wall were all dilated and bled rather freely, but the tumor itself seemed remarkably avascular. The wound was closed with drainage. The tissue removed by biopsy was diagnosed Hodgkin's disease. In less than two hours after the completion of the operation the patient regained consciousness. In less than twenty-four hours much of the edema had disappeared. Three days later, the swelling of the face, neck, and upper extremities had almost completely disappeared. The patient was much improved. It was obvious that relief of pressure on the superior vena cava by decompression was the cause of the remarkable improvement in symptoms. The venous pressure following operation was: right arm 7 cm. of water, left arm 8 cm. of water. The patient was so much improved by

March 29, 1930, that she was discharged. She was readmitted in September, 1930, because a "lump" had developed at the right costal margin. A tumor which involved the costal cartilage of the eighth and ninth ribs was then removed. The histologic diagnosis was again Hodgkin's disease. The patient was discharged in October, 1930, and returned during February, 1931, complaining of dull aching pain in her eyes and in her back, headache and lack of appetite, also of a productive cough. She was emaciated and weak; she lost ground gradually and died June 18, 1931, from generalized Hodgkin's disease. (Fig. 2.)

DISCUSSION

At no time following the decompression operation, up to the time of her death did the patient (Case 2) develop edema of her face and

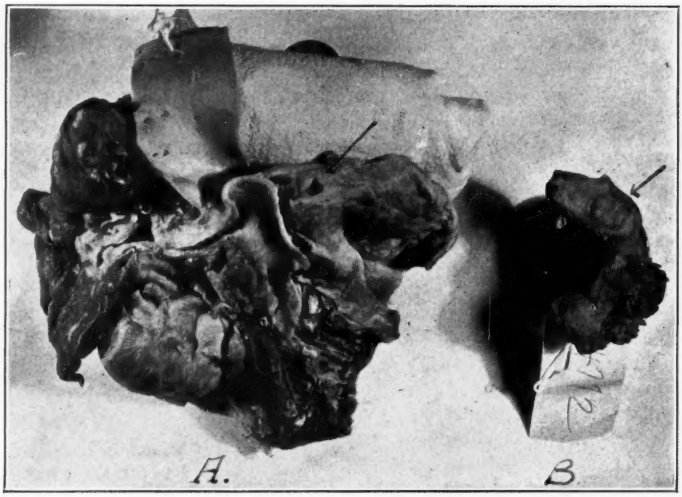

Fig. 2.—(Case 2), courtesy Dr. Howard McCordock. Superior vena cava obstruction due to mediastinal Hodgkin's disease. *A*, the arrow indicates the compressed and narrowed superior vena cava; *B*, the completely obliterated superior vena cava of the patient referred to in Case 1.

arms. Gradually, however, there developed slight external evidence of collateral circulation. Necropsy revealed only a small substernal deeply fibrotic mass which contained tissue typical of Hodgkin's disease. The entire vertebral gutter was lined by similar tissue, and it was also observed about the stomach.

In view of this striking result it is of interest to analyze Fischer's cases and to determine in what proportion of his cases decompression therapy, similar to the procedure followed in this case, might theoretically have been applied with profit.

At the present time it would seem that in only one group might decompression therapy have been considered, inasmuch as in this group only was the important factor of increased intramediastinal

pressure to be considered. In this group are included: (1) aneurysms, (2) malignant, and (3) benign neoplasms. These accounted for approximately 80 per cent of all cases.

In considering cases due to aneurysm it is obvious that with an increase in the size of the aneurysmal sac there is associated an increase in intramediastinal pressure which might result in compression of the superior vena cava. It is reasonable to assume that in some cases decompression therapy should be of distinct benefit by diminishing the pressure exerted by the aneurysm on various structures. On the other hand, such a release of pressure may perhaps in certain cases induce a spontaneous rupture of the aneurysm. Many of the cases of aneurysm are complicated by the presence of inflammatory adhesions. Antiluetic treatment is thus obviously indicated as well. Guleke has performed a decompression operation for aneurysm, with good result.

Considering primary thoracic tumors we find from Fischer's cases that a more or less definite proportion were radiosensitive. From his classification it is difficult to make an exact estimation, but the inclusion of 36 cases of sarcoma (?) suggests the therapeutic possibilities of radiotherapy. Approximately one-third of the primary thoracic tumors were carcinoma. Most of these were pulmonary carcinomas which had invaded the mediastinum. Treatment in these cases could only be palliative. In one-half of these cases there was invasion of the vessel wall and growth into the lumen, and under such conditions decompression would not result in any lasting benefit. However, in the rest of these cases obstruction was due to pressure upon the vein from without. A decompression might have been of distinct benefit in some of these cases.

In the group of cases collected by Fischer, those due to benign tumors would have been aided most. It is certainly wise to remove the tumor whenever possible, but we feel that on some occasions a two-stage operation, the first to consist of a decompression operation, should be considered.

Cases of obstruction due to metastatic growths from extrathoracic tumors are relatively rare. Usually in these cases the immediate prognosis is so grave that surgical intervention would not be justified.

This analysis indicates that in approximately 50 per cent of the 252 cases collected by Fischer, the obstruction was due primarily to compression of the vein. Surgical intervention might have been of value in many of these cases. In another small group of cases, a decompression operation might have prolonged life.

SUMMARY

The literature dealing with obstruction of the superior vena cava, a condition which can hardly be considered as rare, has been reviewed.

Reference has been made to the various forms of treatment which may be employed. Particular attention is drawn to the value of a decompression operation in such cases. A striking result obtained in one instance has been recorded.

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GANGLIONEUROMA OF THE CHEST IN CHILDREN*†

A Report of a Case With a Review of the Literature

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GANGLIONEUROMAS in children are rare if we may judge by the paucity of reports to be found in the literature; and of those which have been described only nine occurred in the chest. Because this condition is curable by surgery and the variable symptomatology may make the diagnosis difficult, it seems worth while to report a case followed for some time before surgery was undertaken and to review briefly our knowledge of the condition and the cases which have previously been described.

Ganglioneuromas are tumors arising usually from the sympathetic nervous system and containing well-differentiated ganglion cells. They were first described by Loretz in 1870. Later it was realized that these tumors bear a close relationship to those formed from undifferentiated sympathetic embryonal cells and which are commonly known as neuroblastomas. These malignant tumors are almost exclusively found in early infancy. Intermediate between these and the true ganglioneuromas are many transitional types. It is our purpose here to discuss only the true, well-differentiated ganglioneuroma which is essentially a benign tumor found uncommonly in childhood and causing symptoms, when present, largely from pressure on adjacent tissues.

Typically this neoplasm is a firm, pale, encapsulated, often nodular mass which grossly resembles a fibromyoma. The tumor may occur singly or in large numbers and may vary greatly in size. Histologically, it is easily recognized with ordinary hematoxylin and eosin stain and is composed of nerve fibers, mostly nonmedullated and arranged in bundles. Ganglion cells occur singly but more often in groups, and multinucleated forms may be seen. It is not unusual to see pigmentation and degeneration of the ganglion cells, and even calcification may be found. The great number of naked axis cylinders in proportion to the number of ganglion cells is usually ascribed to the disappearance of the ganglion cells through degeneration with persistence of the axis cylinders. The tumor typically is almost avascular and exclusive of the nerve elements consists mainly of dense connective tissue and is histologically benign.

There is some evidence that the type of cells in all individual tumors may progress from the undifferentiated sympathetic formative cells

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†Received for publication, July 27, 1923.

which are characteristic of the neuroblastoma to the well-differentiated ganglion cells of the ganglioneuromas. Cushing and Wolbach report a case of a two-year-old child with a tumor which had apparently originated in the region of an intervertebral foramen and had extended into the spinal canal and spinal muscles. On exploration the lesion proved to be a neuroblastoma of sympathetic origin (mistaken at the time for a sarcoma). The growth subsided apparently under the influence of Coley's toxins, but ten years later because of the persistence of paraplegia, a second operation was done which showed "a relic of the former growth whose cells had become completely differentiated into ganglion, capsular and neurilemma cells." Moreover, sometimes both types of neoplasm are found at the same time. Martius reported in 1913 a tumor of the cervical region and posterior mediastinum which was causing marked interference with respiration, and on autopsy the main tumor was found to be a neuroblastoma, but an isolated small tumor nodule was found which was composed entirely of well-differentiated cells and was histologically a true ganglioneuroma. For practical purposes the well-differentiated ganglioneuroma may be considered a benign tumor. In a few instances adult ganglion cells have been found in the regional lymph nodes and have been described as metastatic. It is still a disputed point, however, whether these may not be small individual tumors. At any rate, in cases where such tumors have been completely removed surgically no recurrence or metastasis has been described.

The general impression prevails and is not infrequently expressed in the literature that these tumors are not likely to cause symptoms, but are usually diagnosed accidentally or incidentally at operation or autopsy. A careful study of the ganglioneuromas of the posterior mediastinum which have so far been reported does not bear out this idea.

A summary of the cases previously described follows:

1. Tschistowitch in 1910 described a ganglioneuroma of the right upper mediastinum in a girl of twelve years, found at autopsy in a case of tuberculosis. No history of symptoms or signs referable to the tumor was obtained.

2. Rosenson. 1923. Girl eight years old. Tumor of right upper posterior mediastinum. Patient had persistent cough, slight dyspnea, progressive weakness and loss of weight for one year. Occasional attacks of vomiting and short periods of irregular fever. Malnutrition. Flatness over right chest with diminished breath sounds and absent fremitus and bronchial breathing at angle of scapula. Heart slightly displaced to left. Right palpebral fissure smaller than left. Right pupil smaller than left. Enophthalmus of right eye. (Horner's syndrome.) Operation with recovery.

3. Stout. 1924. Boy two and one-half years old. Tumor of left upper mediastinum. Child had weakness of legs for four months with dragging of right foot. Loss of sphincter control five weeks. Dullness, sibilant and sonorous râles and bronchial breathing over left upper lobe anteriorly and posteriorly. Heart not displaced. Bilateral Kernig and clonus. Sensory changes in legs. Operated on seven months later. Extradural encapsulated tumor found in the spinal canal which was continuous with the tumor mass in chest. Died soon after operation.

4. Crile and Ball. 1929. Girl eight years old. Tumor of left cervical region and mediastinum. Brassy cough and audible breathing for three years. Occasional dyspnea. Anorexia, headache and vomiting. Roentgen therapy for eight months previous to operation. No eye changes. Slight dilatation of veins over upper chest wall. Trachea and esophagus displaced to right. At operation section taken for diagnosis only.

5. Riggs and Good. 1929. Boy four years old. Tumor of right cervical region and mediastinum. Swelling of right cervical region for nineteen months following scarlet fever. Increasing hoarseness and persistent cough. Cyanosis and stertorous breathing. Right pupil larger than left and right pupillary reflex diminished. Trachea displayed to left. Temporary relief from incomplete removal of tumor mass followed by roentgen therapy. Death sixteen months later.

6. Harvey. 1930. Girl nine years old. Tumor of right upper mediastinum. Persistent cough, worse at night, night sweats and lassitude for over a year after bronchopneumonia. Occasional dyspnea and cyanosis. At other times free of these symptoms. Nutrition good. Harsh voice. Marked dullness right upper lobe with diminished breath sounds posteriorly and bronchial breath sounds anteriorly. Displacement of trachea. Diagnostic pneumothorax showed tumor to be extrapleural. Operation with recovery, normal appearance of lung on roentgen films and return of voice to normal.

7. Bischoff. 1931. Sex not given. Patient four years old. Tumor of upper portion of right chest. For several months nocturnal dyspnea with inspiratory and expiratory stridor. Following this, severe attacks of cyanosis and hoarseness which lasted several days. Dullness over left apex. Trachea displaced to right. Slight swelling above sternoclavicular joint when child stood up seen to be continuous with chest shadow. Operation with recovery. No eye signs before operation. Typical Horner's syndrome following operation.

8. Jackson and Babcock. 1931. Girl ten years old. Tumor of right upper mediastinum. Lesion discovered during roentgen examination for a respiratory infection. No cough, no symptoms referable to tumor. Malnutrition. No mention of abnormal physical signs. On operation main tumor densely fixed, small growth removed which was attached over inner surface of rib. Six days later second operation; child died.

9. Bigler and Hoyne. 1932. Boy five years old. Tumor of right mediastinum. Dyspnea on exertion and excitement and attacks of asthma for three years. Extreme cyanosis and dyspnea for two and one-half hours before admission. Intubated with transient relief of symptoms. Marked compression and deviation of trachea to left. Died in five days.

It will be noted that in all the cases except the first and eighth, some symptoms referable to the tumor mass have been present. The clinical picture of a benign tumor of the mediastinum is largely produced by pressure on one or more of the structures adjacent to the neoplasm. Cough, dyspnea and cyanosis are associated with pressure on the trachea and would seem the most constant symptoms. The common physical findings of dullness and diminished breath sounds might be due to pressure on the bronchi, or to the tumor mass itself. Pressure on the esophagus would produce pain or difficulty in swallowing, and these symptoms are not mentioned in this series of cases, but vomiting is mentioned several times and was a definite symptom in our case. No evidence of compression of the veins is mentioned except in Crile and Ball's case where dilatation of the superficial veins of the upper chest

and neck was apparently due to pressure on the superior vena cava, as was true in our case. Symptoms of pressure on the vagus, chiefly slowing of the heart, syncope and difficulty in swallowing, are not described; but the symptoms of stridor, brassy cough and inspiratory dyspnea were due to pressure on the inferior laryngeal nerve, and the so-called Horner's syndrome of inequality of the pupils and palpebral fissures and enophthalmus of the affected side is due to involvement of the sympathetic nerves.

The roentgen pictures of cases of ganglioneuroma of the mediastinum bear close resemblance to each other. The tumor invariably occupies the upper part of the chest. Its edge is sharply circumscribed and convex. There may be displacement of the heart, the trachea, or of the esophagus to the opposite side. In the lateral view the tumor is seen to be in the posterior part of the chest and when pneumothorax is done it is clearly seen that the mass is extrapulmonary.

The differential diagnosis lies between encapsulated effusion, cyst, benign and malignant tumors and involvement of the nodes as in Hodgkin's disease. Chest tap should exclude the first, while a tap with a large needle may give tissue enough for a diagnosis. The slow growth and absence of symptoms, or presence of symptoms referable to pressure only, with no evidence of metastases will usually confirm the diagnosis of benign tumor.

Treatment with roentgen ray was given in Crile and Ball's case for eight months previous to operation without any change in the condition and would not seem advisable if operation is to be attempted because of the increase in fibrosis which may follow and make the surgical removal of the tumor difficult. Surgery apparently offers the only chance for radical cure of this condition.

REPORT OF CASE

L. LaR., girl five years old. First seen in March, 1930, with acute respiratory infection of one week's duration. Signs of pneumonia at right base (dullness, bronchial breathing and râles), also dullness and diminished breathing over right upper lobe. Temperature normal on seventh day. Pneumococcus recovered from sputum. In addition to patch of consolidation at right base, roentgenogram revealed a dense circumscribed mass occupying apex and obliterating the pulmonary marking of the first and second interspace. This picture remained unchanged after the pneumonia had cleared. (Fig. 1.) She was taken out of the hospital at this time but allowed to return to clinic where she was observed for a year. She was a well-nourished and normally developed child, gaining steadily in weight; color good; some difficulty in breathing with a frequent harsh nonproductive cough and wheezing on exertion. These symptoms were said not to have been present before pneumonia but may have been overlooked by the family. Certainly the symptoms increased in intensity during the period of observation. Slight dullness and slightly diminished breath sounds were present over right lung posteriorly to spine of scapula with dullness over right apex anteriorly. Rhonchi frequently heard over both lungs. By January, 1931, dullness posteriorly extended to the level of the fifth dorsal spine and anteriorly to the second rib. Breath sounds slightly diminished with broncho-

vesicular expiration, rhonchi persisted over both chests, cough more frequent and harsher. In July, 1931, occasional vomiting after eating was noted. Cough had increased and was worse in recumbent position. A keratitis of the right eye had existed for one week, possibly secondary to trophic disturbances due to pressure on sympathetic nerves. With treatment the ulcer healed in three weeks leaving only a corneal scar. Physical signs in lungs were unchanged; dilation of superficial veins of right side of neck and chest was noted for first time. Roentgenogram at this time showed tumor definitely increased in size, the mediastinum displaced to the left and a lateral view showed tumor to be in posterior portion of chest. After administration of barium, roentgenogram showed the midportion of the esophagus displaced to left and forward by the tumor mass. Because of the increasing symptoms, operation was advised.

In considering operative treatment of a mediastinal tumor, the surgeon has one of three routes to choose from, viz., anterior route, posterior route, or a long intercostal incision with or without the resection of a rib.

Through the anterior or posterior approach the surgeon can further choose, making his procedure transpleural or extrapleural. Each procedure has its advantages

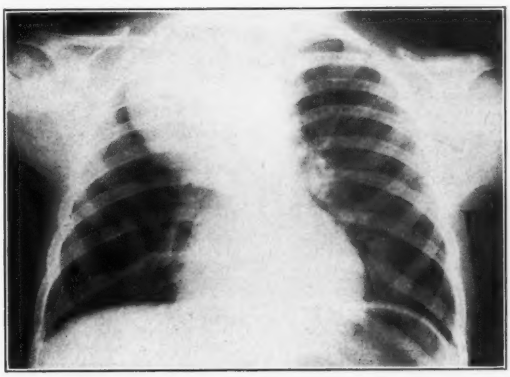

Fig. 1.—Appearance of tumor in roentgenogram.

and its disadvantages. No approach can be selected that will be desirable for all cases; each must be decided individually.

If the transpleural route is chosen, a preliminary pneumothorax has decided advantages. It need not be a complete collapse of the lung, and may preferably be done two weeks before the operative procedure. The advantages are: (1) determination of the presence or absence of adhesions between visceral and parietal pleura; (2) stabilization of the mediastinum; (3) shunting of the pulmonary circulation to the opposite lung; and (4) the resulting compensatory emphysema of the contralateral lung.

The long intercostal incision for a transpleural route is applicable only to cases where there are few or no pleural adhesions. It has the advantages of: (1) rapid approach; (2) good exposure of the tumor; (3) excellent opportunity to explore the pleural cavity; (4) ease of extension upward or downward; (5) rapid and satisfactory closure of wound. The obvious disadvantages are that the entire pleural cavity becomes involved if infection occurs and that accidental hemorrhage is more difficult to control.

The anterior extrapleural route has in its favor an easier external approach, but

more technical difficulties inside the thoracic cage. The pleura is tightly adherent to the costal cartilage and sternum, making it difficult to strip without opening the pleural cavity. In the lower portion the pericardium presents a difficult problem, while nearer the apex the close proximity of the great vessels makes a surgical procedure most difficult.

Obviously, this approach is limited to the removal of tumors of the anterior mediastinum, and even for such tumors it is of doubtful use.

The posterior extrapleural exposure necessitates cutting through the heavy shoulder girdle muscles and reflection of the scapula forward. It requires considerable time to make this exposure and also to repair the wound properly when closing.

The extrapleural routes have the disadvantage of a large denuded area which increases the liability to infection, limits exposure and in which concealed hemorrhage is more liable to occur. It does, however, prevent a surgical collapse of the lung, causes less shock, and if infection should ensue, it does not involve the pleural cavity.

In the case we are reporting the posterior extrapleural route was chosen: (1) because the tumor was definitely in the posterior mediastinum; (2) since it occupied

Fig. 2.—Macroscopic appearance of the tumor.

the apex, we believed it would be found partially extrapleural; (3) the parietal pleura in this region can be very easily stripped from the thoracic cage; (4) if the tumor could not be exposed extrapleurally, this same incision could be converted into a transpleural route.

Operation.—The anesthetic was nitrous oxide and oxygen with a little ether. It was given through a snug fitting intralaryngeal tube, so that intrapulmonary pressure could be increased as desired. A six-inch curved paraspinal incision was made, reflecting the scapula outward. The fifth rib was exposed and four inches were resected subperiosteally. The parietal pleura was then easily stripped beyond the fourth rib and a three-inch portion of this rib was resected. The third rib was divided at the posterior angle and retracted upward. The sixth rib was also divided but not resected. The large hard spherical tumor could now readily be palpated, and its superior pole could be seen projecting above the apical pleura. The tumor was grayish in color, very hard, and the size of a tennis ball. In its retropleural position it had reflected the apical pleura downward, and the parietal pleura having been operatively reflected, it presented a picture having the upper third of the

tumor extrapleural and the lower two-thirds covered by a fold of pleura. The adherent portion of this fold of pleura was easily stripped intact from the tumor.

As there was little or no shock at this stage of the operation, the original plan of doing a two-stage removal was abandoned, and the tumor was removed by finger dissection of its attachment to the posterior chest wall. It was not pedunculated and was firmly adherent to the third dorsal vertebra and rib. This tumor having developed from a sympathetic ganglion was connected to the sympathetic chain, the breaking of which caused severe shock. Venous bleeding was also encountered. Two bleeding points were ligated and the remaining ooze was controlled by a tight gauze pack in the space occupied by the tumor. The chest wall was closed over this pack.

The patient readily recovered from the anesthetic. She was immediately given

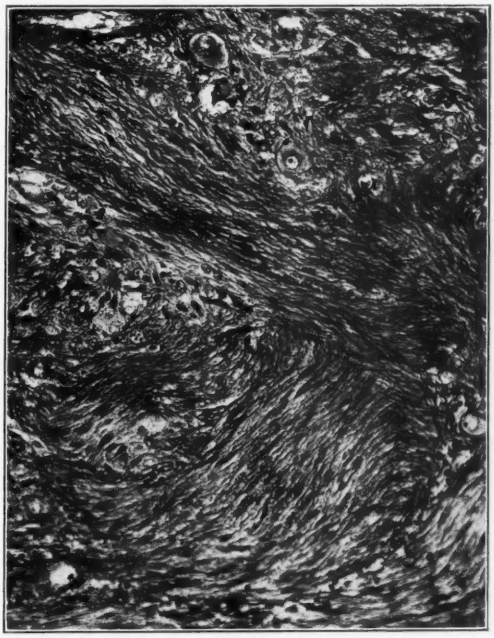

Fig. 3.—A field from a section impregnated according to the Foot and Foot procedure of silver impregnation. This section shows at one side a group of typical ganglion cells and capsule cells; in the middle of the field is a roughly triangular area showing similar cells in a more immature phase.

a transfusion of 150 c.c. of blood and returned to her bed in excellent condition. The entire procedure lasted fifty minutes.

Postoperative hemorrhage occurred at the end of four hours, and the patient died on the way to the operating room for a transfusion and an exploration for hemorrhage.

A postmortem examination through the wound revealed that a ligature had slipped. The cavity was filled with blood. Three small tumors were also found which had not been diagnosed nor found at operation because of their small size. They were lying in the normal position of the sympathetic ganglion of the fourth, fifth, and sixth dorsal vertebrae, the large tumor having been removed from the level of the third dorsal vertebrae.

Pathologic report by Dr. Symmers: Macroscopic examination showed a tumor measuring $7 \times 6 \times 5$ cm. and weighing 110 grams. The tumor was encapsulated and had a grayish white color. The surface was finely nodular. The consistency was firm. On section, the specimen cut with increased resistance and showed a homogeneous structure. The capsule was thin. On one surface, which corresponded to the hilus, an entering nerve trunk 1 mm. in thickness could be seen.

The microscopic picture is well shown in Figs. 3 and 4, for which we are indebted to the Thoracic Tumor Registry to which this case has been submitted.

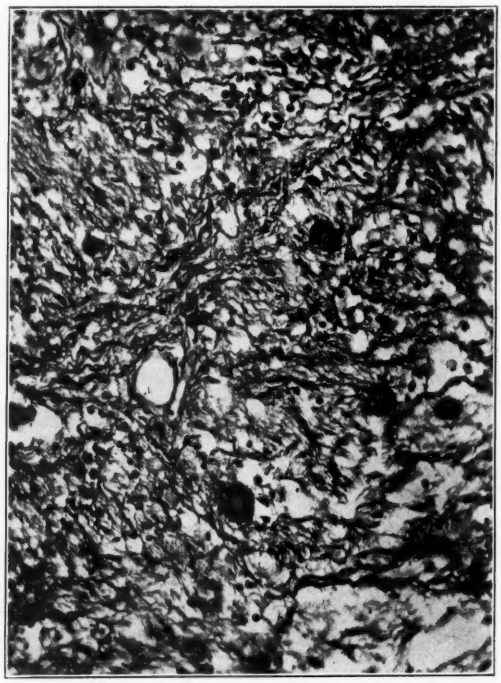

Fig. 4.—A field from a looser area in the same tumor, impregnated by the same method. There is generalized edema, innumerable reticulum fibrils and large ganglion cells.

Our thanks are due to Dr. Lillian Milgram for help in preparation of the bibliography.

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THE ELECTROCARDIOGRAM IN STAB WOUNDS OF THE HEART

A Case Report*†

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FEW cases of stab wounds of the heart have been reported in which the electrocardiographic changes were analyzed. Davenport[1] reported such a case in 1924. The first electrocardiogram, taken nineteen days after operation, showed changes similar to those seen in a recent coronary occlusion. In successive electrocardiograms the changes developed in the usual manner, and abnormalities in the electrocardiograms persisted for as long as eight and a half months after the operation. Bates and Talley[2] reported a similar case. The first electrocardiogram taken five days after the injury showed curves resembling those in recent coronary occlusion. Eighteen weeks after the operation the electrocardiographic curves were again normal. Other cases have been reported by Schlomka,[3] Elkin and Phillips,[4] and Porter and Bigger.[5] Schlomka's case showed changes in the electrocardiogram on the day following the operation such as are seen in recent coronary occlusion. During the following six days the electrocardiographic changes developed in the usual manner. In the two cases reported by Porter and Bigger the electrocardiograms taken nine and fourteen hours after the operation, respectively, showed changes similar to those noted in recent coronary occlusion.

Our case of a stab wound of the heart is reported because an additional opportunity was afforded for following the electrocardiographic changes.

CASE REPORT

On April 14, 1933, L. M., a colored male, aged twenty-two years, was admitted to Michael Reese Hospital. Examination showed a well-nourished, well-developed individual who was suffering from shock. A stab wound one and a half inches long was noted in the left fifth intercostal space just within the nipple line, and two superficial wounds, one in the upper right arm and one in the back of the neck, were observed.

Physical examination and x-ray pictures of the chest led to a diagnosis of stab wound of the heart with hemopericardium, hemo- and pneumothorax.

One hour after admission (8 P.M.) supportive treatment had improved the patient's condition to such an extent that an operation was deemed advisable.

*Aided by the Frederick K. Babson Fund of the Michael Reese Hospital.
†Received for publication, July 31, 1933.
From the Department of Surgery and the Heart Station, Michael Reese Hospital, Chicago.

OPERATION

Under local anesthesia (0.5 per cent novocaine) a cardiorrhaphy was done in fifty minutes. A curved incision, about four inches in length, with convexity downward, was made over the left fifth rib anteriorly. The rib was exposed just above the stab wound; its periosteum was peeled off, and approximately two inches of the rib and cartilage were resected, care being taken not to open the pleura. The muscles were pushed up and retracted. On close examination a small tear was found in the left parietal pleura on its mediastinal aspect, through which a to-and-fro movement of air occurred with each respiration.

A large quantity of dark blood was aspirated from the left thorax. Digital examination disclosed a tear which admitted a finger tip in the parietal pericardium below and medial to the stab wound in the skin. This rent in the pericardium was lengthened to permit examination of the heart. The pericardial sac contained about 500 c.c. of liquid blood which was aspirated. A free oozing of fresh, bright blood into the pericardial sac continued. A wound one and a half inches long was found in the wall of the right ventricle just to the right of the anterior interventricular groove. A clot was found partially to occlude this wound. When this was dislodged, a free stream of bright blood spurted from the wound with each ventricular systole. No other wound in the heart was noted.*

The edges of the wound in the heart were approximated and closed with a double row of interrupted sutures of black silk. This was carried on a full-curved needle because a straight needle will tear the myocardium (Davenport[1]). The first suture was left long to act as a guide for placing the other sutures. The parietal pericardium was closed with interrupted catgut sutures. A flap was used to aid in closing the parietal pleura which was also sutured with interrupted catgut. A silkworm-gut drain was placed in the pericardium and brought out through the skin incision. The muscle and fascia were closed with interrupted catgut suture and the skin with silkworm gut.

The blood loss during the operation was estimated as 1,500 to 2,000 c.c. The patient was given 500 c.c. of normal saline intravenously during the operation and 300 c.c. of blood immediately after. An electrocardiogram was taken during the operation and one immediately after. On the patient's return to the ward the blood pressure was 105/70. A shock enema was given and two hours later a hypodermoclysis of 1,000 c.c. of normal saline with 2.5 per cent glucose.

*At this stage of the operation the patient again passed into a stage of shock. The peripheral pulse was imperceptible, and the heart was seen to be beating feebly. Adrenalin and caffeine hypodermically proved inadequate, so adrenalin was injected into the right ventricular wall and the heart massaged manually with each systole. In a few minutes following this procedure a marked improvement in the circulation became apparent.

PROGRESS NOTES

From April 15 to April 21 the patient's recovery was rather uneventful. He was given supportive treatment and responded very well. Small doses of digalen were also administered. The physical examination of the chest on April 17 and x-ray examination of the chest on the same date showed an accumulation of fluid in the left chest and a markedly enlarged cardiac area. On April 21, 400 c.c. of blood were removed from the left chest. On April 23, 430 c.c. blood transfusion was given. From April 23 to May 16 the patient's recovery was rapid. The cardiac condition was checked routinely by physical examination and electrocardiogram. X-ray examination and physical examination showed a decrease in the size of the cardiac area. At the time of discharge the cardiac shadow approximated normal, the heart tones were distant, and no murmurs or friction rubs were heard.

COMMENT

In Fig. 1 are assembled segments of some of the electrocardiograms taken on this patient. The electrocardiogram taken before operation and three hours after the stab wound (*A*) is not normal, nor is it typical of the curves seen in recent coronary occlusion. The QRS complex is prolonged (0.12 sec.) and slurred in all leads; it is small in Lead III. The S-T segment is slightly negative in Lead III and the T-wave in this lead is small and inverted. The P-R interval is within normal limits (0.12 sec.). Without a control curve it is impossible to decide whether the changes in the electrocardiogram, suggesting intraventricular block, were produced by the stab wound or were present before the injury. However, the fact that the curve taken three months later (*G*) had a similar contour, suggests that the patient had the changes before the stab wound.

Immediately after the operation a striking change in the electrocardiogram is seen (*B*). This consists essentially in a noticeable elevation of the S-T segment in all three leads with partial obscuring of the T-wave. Thus, in Lead II the S-wave is really above the isoelectric line, the S-T segment in Leads I and II rises to fuse with the T-wave, and the S-T segment in Lead III is a descending one. A small Q-wave has appeared in Lead III, the QRS complex remains prolonged, and the P-R interval is unchanged.

During the course of the next month the S-T segment became horizontal and less positive and eventually isoelectric. The T-wave in Leads I and II became smaller, and a negative T-wave reappeared in Lead III. The Q-wave in Lead III tended to disappear. The S-wave returned to below the isoelectric level. Later (*E*) a small negative T-wave appeared in Lead II which became diphasic (*F*) and eventually upright (*G*).

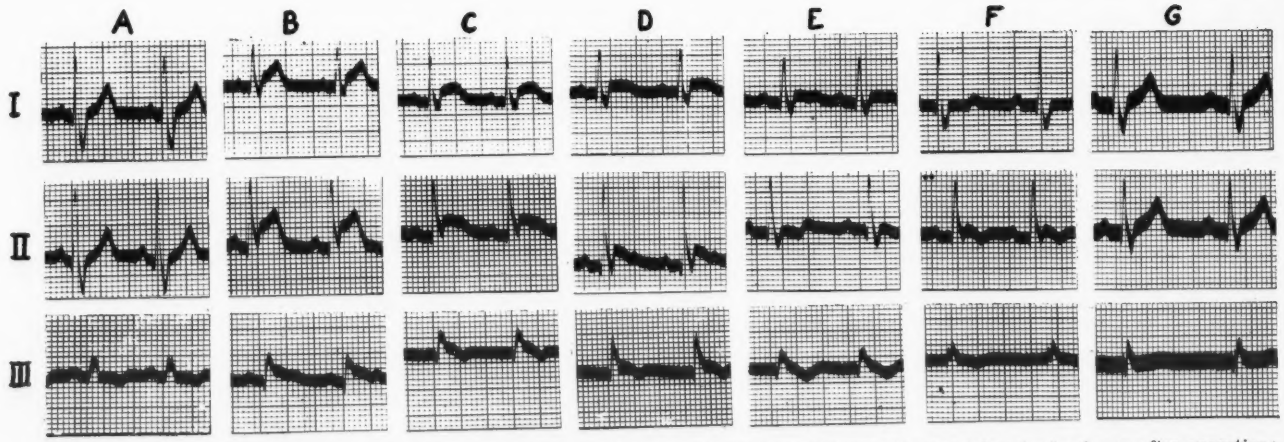

Fig. 1.—*A*, Segments of electrocardiogram taken before operation; *B*, segments of electrocardiogram taken twelve hours after operation; *C*, segments of electrocardiagram taken thirty-six hours after operation; *D*, *E*, and *F*, segments of electrocardiogram taken two, three and six days after operation; and *G*, segments taken three months after operation.

The curve taken three months after the operation resembles that immediately after the stab wound except that the T-wave in Lead III has become positive.

The deviations in the electrocardiogram in this case resemble the type of changes reported in pericardial effusion in man (Scott, Feil and Katz[6]) and in the dog (Katz, Feil and Scott[7]), more than the changes seen in recent coronary occlusion. It is not common to find an elevation of the S-T segment in all three leads in recent coronary occlusion (cf. Bohning and Katz[8]). There is x-ray evidence of enlargement of the cardiac shadow in this case which waxed and waned more or less parallel with the deviations of the electrocardiogram; and a similar parallellism is seen in the circulatory condition of the patient. Both the x-ray and clinical findings could be explained by the temporary appearance of a pericardial effusion. It is not necessary to assume a large pericardial effusion because, as Katz and Wallace[9] have shown, a large dilated heart with a small effusion will produce a similar effect. The hemorrhage may have come from oozing from the heart wound or from seepage from a lung tear.

There is no doubt that a small branch of the left anterior descending coronary artery was ligated in the operation. It is doubtful, however, whether this small occlusion by itself could produce as extensive a series of changes in the electrocardiogram as were seen in this case. Smith and his coworkers[10] have recently shown that ligation of small branches of the left anterior descending coronary arteries leads to very little change in the electrocardiograms in dogs. The ligation of this small coronary branch may have helped to cause the dilatation of the heart; but it is a minor factor compared with the injury resulting from the suturing and extensive handling of the heart, which was sufficient during the operation to lead to shock.

The electrocardiographic changes can be interpreted in this case on the assumption that the ischemia of the heart was brought about by a tamponade effect of fluid in the pericardium, perhaps small in amount but acting on a greatly dilated heart. It is possible that a similar tamponade action may explain in part the electrocardiograms reported by other observers following repair of stab wounds of the heart.

SUMMARY

A case is reported in which serial electrocardiograms were taken after a stab wound of the heart and following its surgical repair. In this case the changes in the electrocardiogram resemble those seen following acute pericardial effusion, and are therefore attributed to the tamponade action of a pericardial effusion on a dilated heart.

We wish to thank Dr. Louis N. Katz for his valuable suggestions and criticisms.

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TUBERCULOSIS OF THE THORACIC WALL*

A Survey of 32 Cases

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ALTHOUGH tuberculosis of the thoracic wall is in itself usually a condition of little gravity, it is surprisingly resistant to treatment. A small cold abscess, beneath which roentgenograms may show no lesion of bone or cartilage, may be the superficial expression of widely extending tuberculous sinuses and tuberculous involvement of several ribs and cartilages. Repeated aspirations of the abscess or incomplete or even presumably complete surgical removal of the grossly involved tissues may result in apparent healing of the lesions, but it is notorious that early or late recurrence of the disease is frequent and that repeated operations may be required to effect a lasting cure. Obviously, the extent and severity of the thoracic wall lesions and of the associated tuberculous lesions of the pleura, lung or other organs have an important bearing upon the success of treatment.

Tuberculosis may involve any structure in the thoracic wall. The cartilages and anterior portions of the ribs and especially the regions of the costochondral and chondrosternal junctions are most frequently involved. The posterior or lateral portions of the ribs and the vertebrae are less often the source of abscess of the thoracic wall. The most frequently diseased soft tissues are the pleurae, the mediastinal, internal mammary and intercostal lymph glands, muscle, breast, skin and the extrapleural, submuscular and intermuscular tissue planes. It is unusual for an abscess of the thoracic wall to rupture into the pleural cavity or lung.

In the great majority of cases more than one tissue element is diseased at the time that the diagnosis is made, and one or more sinuses are often present. Among our 32 patients, there was a cold abscess involving only the soft parts in 10, two of them having two abscesses each. Eight had, in addition to soft tissue abscesses, lesions of a single rib, cartilage or the sternum; 5 patients had lesions of two ribs or cartilages, and 9 had two or more separate lesions variously involving ribs, cartilages, spine, sternum and clavicle. The more thoroughly an apparently simple cold abscess is explored, the more frequently sinuses will be found leading to diseased cartilage or bone. The failure of roentgenograms to reveal lesions of cartilage and some-

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Received for publication Feb. 12, 1934.

times of sternum or rib tends to cause the physician to underestimate the extent of the infection.

ETIOLOGY

Much has been written about the etiology of tuberculosis of the thoracic wall, but there is no consensus of opinion about the routes of infection. Some believe that tubercle bacilli are carried by the blood into the ribs; this is probably true in those occasional cases in which the apparently oldest lesion is a costal sequestrum. It is certain that in some cases the infection is due to direct extension from tuberculous axillary or mammary glands or from a tuberculous empyema, especially if it has been aspirated or drained. We are not considering in this study those benign cases of thoracic wall tuberculosis which consist of a mild, strictly localized tuberculous infection, perhaps complicated by a tiny abscess, which occasionally follows aspiration of a tuberculous empyema.

It is highly probable that the infection in many cases of thoracic wall tuberculosis passes from a tuberculous lung or pleura through the lymph vessels to the internal mammary or intercostal lymph glands or to the costal or sternal periosteum or to the perichondrium and from there into rib, sternum, cartilage and soft parts. Whether secondarily or primarily involved, the ribs and cartilages are infected in the majority of patients at the time that they are seen by a surgeon.

In 5 of our 32 patients there was no evidence of tuberculosis except in the thoracic wall; in one of these there was a history of local injury. In 9 other cases there was no demonstrable direct connection between the thoracic wall tuberculosis and the known tuberculosis of the lung, distant bone, peritoneum, salpinx or anus. In 7 cases there was an associated tuberculous empyema, 4 of which had been drained and 2 others had extensively infected needle tracts; 1 empyema was healed at the time of the appearance of the lesion in the fourth to seventh cartilages, but the axillary glands were actively tuberculous at the time. Five extrapleural thoracoplasty wounds became infected with tuberculosis. Two of these operations were for pulmonary tuberculosis (the pleura was not known to have been injured) and three more for tuberculous empyema, 1 having an infected needle tract. It is not rare to find small lymph glands in the intercostal spaces during extrapleural thoracoplasty operations for phthisis without empyema, and injury to them or to the lymph vessels while stripping the periosteum from the ribs may infect the wound with tubercle bacilli. In 2 cases there was an associated pleural effusion without needle tract infection, and in 4 cases tuberculous glands were found in the field of operation for the thoracic wall lesion; 2 of these 4 had active pulmonary tuberculosis, 1 had inactive pulmonary tuberculosis and 1 had no other demonstrable tuberculosis. In summary,

among the 32 cases, associated pulmonary tuberculosis was active in 15 cases and inactive in 8; there was tuberculous empyema in 11 (4 of which had been drained); pleural effusion in 2; tuberculous adenitis in the operative field in 4 and at a distance in 3; tuberculous needle tract infection in 5; and no demonstrable direct source of infection in 16, 5 of these having no known tuberculosis except in the thoracic wall.

An analysis of our cases suggests that there is no direct relationship between the severity and the extensiveness of the thoracic wall lesions and those in the lung, pleura and other organs. In fact, among the 14 patients having the most extensive involvement of the thoracic wall, 4 had no other demonstrable tuberculosis, and 2 had only healed pulmonary tuberculosis, whereas among the 10 patients with lesions of only the soft parts of the thoracic wall, 1 had a huge tuberculous mediastinal abscess and 8 had active pulmonary tuberculosis (4 having in addition tuberculous empyema, 2 laryngeal tuberculosis and 1 osseous tuberculosis).

DIAGNOSIS

The diagnosis of tuberculosis may be tentatively assumed when a painless or only slightly painful swelling or abscess or sinus that does not tend to heal forms in the thoracic wall, especially if there is tuberculosis elsewhere in the body.

All of our 32 patients were proved to be tuberculous by demonstrating tubercle bacilli in aspirated pus or in wound secretion by smear, culture, guinea pig inoculation or by finding typical tubercles in a specimen of the diseased tissue. In 10 other cases in which the clinical diagnosis was tuberculosis, this was not proved by laboratory examination and so these cases are not included in our study.

Our experience with regard to the laboratory examinations of the 32 proved cases follows: Twenty smears were taken from 13 patients and only 3 were positive for tubercle bacilli; the 10 patients whose smears were negative had tubercles in granulations removed from the mouth of a sinus or in the diseased tissue removed at operation; 3 of the 10 were positive, and 1 was negative to guinea pig inoculation; and 1 was negative to culture. Three specimens from three patients were cultured for tubercle bacilli; 2 were positive, 1 of these also having a positive smear and 1 a positive tissue examination; the negative culture case also had a negative smear but positive tissue specimens. Seven of 9 guinea pig inoculations from 8 patients were positive, one patient being positive only on the second examination. The patient giving a negative guinea pig reaction had a positive tissue examination. Among those having positive guinea pig inoculations, 3 had from one to three negative tissue examinations each, 2 had one or two negative smears and one positive tissue examination;

1 had four negative smears and two positive tissue examinations; 1 had two negative tissue examinations and a positive smear and a positive culture. In 31 patients diseased tissue was examined microscopically and 28 were positive, 24 on the first examination, 1 on the second and 3 on the third. Three patients had from one to three negative examinations. The single patient who had no tissue examination was positive to smear and culture. The three patients negative to tissue examination were all positive to guinea pig inoculation, 1 being also positive on concentrated smear of the pus. Among 10 of the patients with positive tissue examinations, 8 had from one to four negative smears; 1 had two negative smears and a negative guinea pig inoculation; 1 had both a negative smear and a negative culture.

Ninety per cent of the patients who had tissue examinations were positive after one or more examinations (77 per cent at the first examination). Eighty-eight per cent of those having guinea pig inoculations were positive (75 per cent at the first examination). Twenty-three per cent of those having smears were positive (15 per cent at the first examination). The conclusion is apparent that the diagnosis of tuberculosis may not be excluded because of a single negative examination by any one method of laboratory examination but that the use of repeated examinations and of multiple methods of examination is highly efficient. In this connection it should be mentioned that 7 of the 10 presumably tuberculous patients that are not included in this study had only negative tissue examinations and the remaining 3 had no laboratory examination whatever. If the 7 patients who had negative tissue examinations had tuberculosis, the efficiency of laboratory methods of diagnosis, at least in the unsystematic way in which we employed them, is only 82 per cent efficient. Roentgenology is of diagnostic value when lesions are visible; tuberculous lesions of cartilage are not demonstrable in roentgenograms. Thickening of the pleurae may be seen. Roentgenograms revealed evidence of osseous tuberculosis in 14 of the 19 patients for whom roentgenology was used.

Eighteen of our 32 patients were between ten and thirty years of age, 2 were from one to ten years old, and 4 were more than fifty years.

Differential diagnosis is chiefly concerned with syphilis, nontuberculous (including typhoidal) osteomyelitis or abscess, empyema necessitatis, actinomycosis, blastomycosis and any solid or cystic neoplasm involving the thoracic wall. Syphilis must be especially suspected if there is a tumor involving the upper sternum or sternoclavicular region. A tuberculous thoracic wall abscess may have such a thick wall of infiltration that it may feel like a solid tumor; in one such case we were suspicious of carcinoma of the breast until operation revealed the true nature of the lesion.

TREATMENT AND RESULTS

The apparent triviality of a thoracic wall abscess frequently leads the surgeon merely to aspirate and inject it or to drain it and perhaps to curette, cut or cauterize away the most obviously necrotic cartilage or bone. Although occasional patients are cured by these methods, especially when bone or cartilage is not necrotic, most surgeons who have had a wide experience with the disease agree that such measures may be expected to be futile. Radical excision of every bit of infected tissue and of any normal cartilage that has been cut into, and suture of the wound with or without drainage, rather than open packing, is the surgical treatment that may be counted on to give the best ultimate results. Unfortunately, this ideal operation frequently cannot be performed, either because the patient is too ill with pulmonary or other tuberculosis or because complete excision of the lesions would require partial removal of structures such as the diaphragm, the pleurae or pericardium, or would cause too great exposure of the heart from removal of much of the sternum and many cartilages. Occasionally it is best merely to aspirate or, if the abscess is secondarily infected, to drain, and later, if the patient's condition improves sufficiently, to perform a radical operation.

Radical resection of all tuberculous tissue before spontaneous fistulization of the abscess has occurred, and when the local and general conditions permit, is strongly recommended by König, Moschcowitz, Ito, ourselves and others. Moschcowitz insists upon the additional resection of undiseased cartilage whose perichondrium may have been even slightly damaged during the operation. He also insists upon the complete removal of any cartilage, even if apparently undiseased, that has been cut across during the operation. He further states that because of the contiguity of the sixth to tenth cartilages, inclusive, through their interchondral articulations, all of these cartilages, as well as the uncalcified ensiform process of young persons, must be completely removed if a portion of any one of these cartilages is diseased or has been cut into. His position is based upon the frequent relapses that occur when any cartilage is left exposed in the infected wound. Moschcowitz reports five cases, two of which became completely healed. König, in the best treatise that we know on the subject of tuberculosis of the thoracic wall, reported in 1906 upon 110 operated cases. Two patients died in the hospital of miliary tuberculosis, 1 of widespread tuberculosis and 1 of pneumonia. The thoracic wall tuberculosis became healed in 73 of the remaining 106 patients. Eighty-four of these 106 had radical operations and 62 became healed; whereas 22 were operated on with a sharp curette, and only 11 became healed. Sixty-six of the 110 patients had tuberculosis elsewhere than in the thoracic wall. In 50 it was in the lungs, in 20 in the

bones, and some of these also had pulmonary tuberculosis. A considerable number of patients whose thoracic wall tuberculosis became healed later died of pulmonary tuberculosis. Ito reports upon 31 patients, for all of whom he apparently found it possible to eradicate the visible lesions radically. It would appear from his report that all the lesions became healed, though his statement is indefinite about 7 of the patients.

In only 7 of our patients did we resect virtually all the diseased tissue and remove all normal portions of cartilages that had been cut across in an infected field. Six of the 7 are healed; 4 healed after the first operation and 1 after the second. One patient's wound did not close and later he died of a "stroke." The last patient's wound is now healed four months after his third operation; the wound had remained healed for five months after his second operation. The wound of one of the healed cases closed, opened and then closed again. Five of the healed patients were the ones of this group who had surgical suture of their wounds, with or without drainage; three of these wounds opened and then closed. The apparent cause of reopening of a wound and of the failure of two wounds to close was an infected rib stump. The thoracic wall lesion in the 6 healed cases was cold abscess only in 1, and tuberculosis of the soft tissues and one rib or cartilage in the others. The one unhealed patient had three lesions, the one considered in this group involving soft tissues and three ribs. Four of the 7 patients in this group had active pulmonary tuberculosis; the thoracic wall tuberculosis is healed in all, and the pulmonary lesions are apparently arrested, arrested, or quiescent; the fourth patient, discharged from the hospital five and a half years ago, has been lost track of. Three patients had only the scars of healed pulmonary tuberculosis, and the wounds of two are closed and one was open. Before we saw the 7 patients of this group, 4 had been surgically drained, and 1 had had aspirations and roentgen ray therapy and a spontaneous sinus had formed; all of them were secondarily infected.

Another group of our patients consists of 7 in whom radical resection of the lesions was performed as in the first group, except that the apparently normal portion of one or more sectioned cartilages was not removed. The wounds of only 3 of these patients are healed, 1 of them after a second operation. Five patients had surgical suture of the wound with or without drainage but all reopened. The 4 unhealed patients did not return for secondary operation. The lesions of this group were somewhat more extensive than those of the first group. Only 2 patients had a single diseased cartilage, rib or sternum; 1 of them is healed. Three had disease of two ribs or cartilages each, and none is healed. Two, having had involvement of four cartilages of the costal arch, are healed. In one of the healed cases

the patient had active pulmonary tuberculosis and a tuberculous spondylitis, for which she had a surgical spinal fusion and spent eighteen months on a Bradford frame; all lesions are healed and she is working. Two patients had inactive pulmonary tuberculosis; the thoracic wall wound of one is healed and of one not healed. One of the 4 patients who had no evidence of pulmonary tuberculosis is healed. Five of the 7 patients in this group had had their thoracic wall abscesses drained before we saw them. The 3 healed cases are included among the 4 which had prolonged courses of ultraviolet light treatment.

In 9 patients the operation was even less complete than in the preceding group, in that some infected soft part or skeletal structure was not removed, usually because of the extensiveness of the lesions. These 9 patients had sixteen operations, and only 2 are healed, one after the first operation and the other after the second operation. König reports 50 per cent of 22 patients healed after the use of a sharp curette, whereas 73.8 per cent of 84 patients became healed after radical removal of all diseased tissue. Of 4 unhealed cases, 5 had one operation, 1 had two operations and 1 seven operations. The thoracic wall lesions in the 9 patients were of the soft tissues only in 2 patients (1 healed); soft tissues and mediastinal abscess from the fourth to the eleventh vertebral bodies and nontuberculous osteomyelitis of three ribs (all lesions healed); 3 unhealed patients had draining tuberculous empyema and involvement of the soft tissues and from one to three ribs; 1 unhealed patient had tuberculous involvement of the soft tissues, four ribs and four cartilages and an empyema which healed under treatment; 2 unhealed patients had multiple, distinct thoracic wall lesions variously involving several ribs, cartilages, sternum, and clavicle. Except in the cases with involvement of only the soft parts, the extensiveness of the lesions was the reason for their incomplete resection. There was active pulmonary tuberculosis in 3 cases; the wound of one is healed and the pulmonary disease is improved; the pulmonary disease of the second patient is apparently arrested after a thoracoplasty, but the incision is infected with tuberculosis and unhealed; the third patient had both active pulmonary tuberculosis and a pyogenic and tuberculous empyema, and he died five weeks after a Schede thoracoplasty with an unhealed wound, and at necropsy tuberculosis was found in the liver, spleen, and kidneys. Only 1 of the 5 patients with no demonstrable pulmonary tuberculosis has a healed wound; one died five weeks after operation on the thoracic wall tuberculosis from nontuberculous pulmonary abscesses and empyema, and one died two years later from generalized tuberculosis and a tuberculous empyema. Before we saw the 9 patients of this group, 2 had had drainage of their thoracic wall abscesses and ultraviolet light treatment; 4 had had drainage of

tuberculous empyemas; and 3 had had no local treatment, our 2 healed cases in this group being among these 3. After our operations, 4 patients were given ultraviolet light treatments and none became healed.

In 5 patients incision and drainage was the only operation performed, one patient having had two operations, and, in addition, one had been inadequately drained before admission. None is healed. In 3 patients the general condition was too poor to permit a more extensive operation than mere drainage; the extent of their thoracic wall lesions is not known. In the remaining 2 patients, the tuberculous infection of the thoracic wall followed extrapleural thoracoplasty operations, and simple drainage was believed to be adequate treatment for them. Four patients had active pulmonary tuberculosis, for which extrapleural thoracoplasty was performed in 3, in 2 of whom the sputum is negative and the general condition improved. The third thoracoplasty patient died nine months after operation from tuberculosis of the kidneys, lungs, intestines, larynx, and thoracic wall; the thoracoplasty incision had become tuberculous and a separate subpectoral tuberculous abscess developed, both lesions requiring drainage. One patient in this group had no demonstrable pulmonary tuberculosis but had spinal and other osseous tuberculosis and died of tuberculous meningitis. Two patients in this group had ultraviolet light treatment after operation.

In only 1 of our 32 patients was aspiration alone performed. Subsequent excision was contemplated, but the abscess did not recur, nor had it at the time of our follow-up report fourteen months after aspiration. This patient also had active pulmonary tuberculosis and a pure tuberculous empyema; a thoracoplasty was performed and all the tuberculous lesions are apparently arrested.

We are not prepared to offer an opinion as to whether simple aspirations, heliotherapy and prolonged bed rest, and perhaps roentgen ray therapy, are better treatment than is radical surgical resection for the apparently localized type of early lesions. Our experience includes relatively few of these early, simple cases. It is the experience of some sanatorium physicians that many such patients apparently become cured after prolonged bed rest and aspirations of pus, if present. This is in conformity with the good results from nonsurgical treatment of tuberculous adenitis, but good results are less likely to occur if the tuberculous infection involves cartilage or bone as well as the soft tissues.

In 4 of our patients no surgery was used by us; none became healed. In 2 of them the local and general tuberculosis was too extensive for operation; in another patient the thoracic wall lesions were extensive, diagnostic aspiration was performed once, but no reason appears on

the history as to why surgery was not used; the fourth patient refused operation. All these patients had multiple lesions of the thoracic wall, variously involving ribs, sternum and clavicle; three of them had draining sinuses at the time of admission. Two of the four patients had active pulmonary tuberculosis, and one other had tuberculous salpingitis, peritonitis, and fistula in ano. König cautions against relying entirely upon general antituberculosis measures, important though they be. He recommends radical resection of the lesions before an abscess spontaneously fistulates, when mixed infection and extension of the tuberculous lesions are to be expected. Hans Iselin in 1919 strongly recommended roentgen ray therapy without surgery. Forty of his 57 patients treated from 1909 to 1915 are healed; 7 did not heal and died of pulmonary or osseous tuberculosis. From 1915 to 1917, 20 of 28 patients were treated by roentgen rays and 16 of these 20 are healed, 2 are unimproved and 2 died without having become healed. His 72.7 per cent of healed cases is surprisingly high in view of the fact that no necrotic cartilage or rib was removed surgically. In many of our 32 patients more or less prolonged courses of ultraviolet light treatment were given postoperatively, and we feel that these patients did better than those who did not receive it, but we were often disappointed to find that heliotherapy was unable to bring about closure of an open tuberculous wound. All patients should be placed upon a carefully regulated antituberculosis regimen.

Summarizing the results in our 32 patients, only 12, or 37.5 per cent, have healed thoracic wall tuberculosis. All of them are subject to recurrence. The operations have been performed during the last eight years, and all but 3 of the 32 patients have recently been traced by letter or medical examination. In the group of 7 patients with radical excision of the lesions and removal of any normal cartilage that had been sectioned, there were 10 operations and 6 cures (86 per cent for the patients and 60 per cent for the operations). In the group of 7 patients with radical resection but without removal of sectioned normal cartilages, there were 9 operations and 3 cures (44 per cent for the patients and 33 per cent for the operations). In the group of 9 patients with incomplete excision of the tuberculous lesions, there were 16 operations and 2 cures (22 per cent for the patients and 13 per cent for the operations). In the group of 5 patients with mere incision and drainage there were 6 operations and no cures. Only one patient was treated by aspiration only and he is cured. In the group of 4 patients with no treatment except ultraviolet light, there was no cure. We are at a loss to understand why our results should be so much inferior to those reported by König, Hans Iselin, and Ito. A possible explanation is that our patients, mostly referred from sanatoriums where the simple cases with relatively favorable prognosis had been cured by nonsurgical measures,

may have averaged a higher proportion of extensive and grave tuberculous lesions wide of the thoracic wall. In only 5 of our 32 patients was the thoracic wall tuberculosis the only tuberculosis in the body; in 3 others there was evidence of only healed pulmonary tuberculosis. The remaining 24 patients had complicating single or multiple and more or less grave active tuberculous lesions in various systems of organs. Another explanation for our relatively poor results is that our patients were followed for from four months to eight years, whereas the longest of König's and Ito's observations were approximately three years; the longer the patients are followed, the more likely are recurrences to be found.

Our best results were in the 5 patients who had complete removal of the lesions and suture of the wound with or without temporary drainage but without packing. These 5 patients had 6 operations and all are healed. Three of them had pure tuberculous infection, and 2 had pyogenic and tuberculous infection. Only 1 of 6 patients (8 operations) whose wounds were sutured after incomplete excision became healed. The average time for closure of the wound was much less when the surgical incision was sutured than when it was not.

A survey of the 12 patients whose wounds are healed is of interest. The patient who had no other tuberculous lesion is doing full-time work two and a quarter years after operation. One patient with no other tuberculosis, but with diabetes, is doing full-time work six months after operation. A patient with healed pulmonary tuberculosis and tuberculous empyema is doing full-time work four and a quarter years after operation. One who had a huge mediastinal abscess is able to work three years after operation. One who had active pulmonary and spinal tuberculosis is doing full-time work two years after operation. One who had active pulmonary tuberculosis and pure tuberculous empyema, which was treated by phrenic nerve interruption, aspiration, and induced pneumothorax, is doing full-time work two and a quarter years after operation; another patient who had the same complications has had a thoracoplasty and is convalescing in a sanatorium one year after operation; another patient with the same complications had no direct treatment for them and has been lost track of. Three patients with complicating active pulmonary tuberculosis have had, respectively, thoracoplasty, phrenic nerve interruption, and sanatorium regimen alone (childhood type of tuberculosis), and all are now convalescing in sanatoriums ten, nineteen and five months, respectively, after the operations on their thoracic wall tuberculosis. None of the 12 patients has tubercle bacilli in the sputum, if present; all are afebrile and 3 of the 4 patients who are still in sanatoriums are taking exercise, the fourth having been there only five months.

Seven or 22 per cent of the 32 patients are dead. We have been unable to trace three patients, and it is possible that they are dead. All of the 7 patients had had serious complications of their thoracic wall tuberculosis at the time of admission to the hospital. All had involvement of multiple structures in the thoracic wall, and 6 had two or more separate thoracic wall lesions. None had a healed wound at the time of death. The general condition of 3 patients was so poor that no more extensive operation than drainage could be performed. In 2 other patients the lesions were too extensive for complete excision. The causes of death in the 7 patients were: (1) miliary tuberculosis one month after a Schede thoracoplasty and sixteen months after excision of the tuberculous thoracic wall lesions and drainage of a pyogenic and tuberculous empyema; (2) and (3) pulmonary and generalized tuberculosis nine months and two years, respectively, after discharge from the hospital; (4) pulmonary tuberculosis and pyogenic and tuberculous empyema nine days after drainage of the thoracic wall abscess (the patient was desperately ill at the time of admission); (5) tuberculous meningitis five weeks after drainage of the abscess; (6) nontuberculous suppurative pneumonitis, pulmonary abscess, and empyema, which preceded the signs of the thoracic wall tuberculosis (death occurred five weeks after drainage of the empyema); (7) a "stroke" two years and two months after apparently complete removal of the thoracic wall tuberculous lesions.

The only nontuberculous postoperative complications were two cases of acute otitis media, occurring within two weeks of operation.

SUMMARY AND CONCLUSIONS

1. Tuberculosis of the thoracic wall is in itself a condition of little gravity, but it is surprisingly resistant to treatment. A simple cold abscess may be the superficial expression of extensive tuberculous involvement of the sternum, cartilages, ribs, and soft tissues. The thoracic wall lesions are often only a part of multiple tuberculous lesions in different parts of the body.

2. The thoracic wall may become infected through lymph channels from active tuberculous lesions in the lung, mediastinum, pleura, or neighboring glands. Or it may become infected through the blood stream or by direct continuity with a draining tuberculous empyema or tuberculous glands.

3. Of the laboratory examinations, microscopic examination of the diseased tissue and guinea pig inoculation and culture of pus from an abscess or sinus are the most efficient. Examination of smears of pus is relatively inefficient. Roentgenograms do not show tuberculous disease of the cartilages and occasionally they fail to show lesions of the sternum and ribs.

4. Our best results were obtained after complete resection of all involved soft and skeletal tissue, including the undiseased portions of any sectioned cartilages, and complete suture of the wound, with or without temporary drainage.

5. Less good results were obtained when treatment was less radical. However, the lesions in these patients tended to be more extensive than in the patients whose lesions were completely removed. It may be that nonsurgical treatment by aspirations, heliotherapy, prolonged bed rest and perhaps roentgen rays is better for early lesions than surgical excision. Our series includes few early cases with limited lesions.

6. Only 12, or 37.5 per cent, of our 32 patients have completely healed wounds. Seven, or 21.9 per cent, are dead. These poor results are in part due to the fact that the great majority of the patients referred to us had extensive and often multiple lesions of the thoracic wall and complicating tuberculous empyema and active tuberculosis of the lungs or other organs.

7. Repeated operations are often required to eradicate newly infected rib, cartilage, or sternum. Even if the wound has healed solidly, recurrences of active tuberculous infection are not unusual. We believe that the difficulty of completely and permanently curing any but the simple cases of thoracic wall tuberculosis has not been sufficiently stressed in medical writings.

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FACTORS INFLUENCING THE SAFETY AND EFFICIENCY OF THORACOPLASTY*†

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ONE of the early objections to the performance of extrapleural paravertebral thoracoplasty was the high operative mortality. More than a quarter of a century of clinical experience has dispelled much of this criticism, but there still remain certain factors responsible for complications. It is only through a careful study of details that these may be reduced to a minimum. A few of them will be mentioned.

POSITION ON THE OPERATING TABLE

Different positions on the operating table have been selected for patients undergoing thoracoplasty. The sitting position has been used, as well as the one in which the patient lies on the side with the involved lung uppermost. Each of these two positions has definite advantages, the chief one being the ease of adequate exposure and the accessibility of the first rib. The difficulty of the anesthetist is reduced to a minimum.

One important factor has been overlooked. Every one doing this work is familiar with the dangers of spreading the tuberculous infection and aspiration pneumonia. In using either of these positions this one danger is a constant menace and conducive to these most feared complications. It occurred to us that any position which could reduce this danger would be well worth while unless this change would in turn produce some deleterious effect. The prone position is the only other one to be considered. The question immediately arising was, what would happen to the vital capacity of an individual when assuming this position. If it remained equal to that recorded in other positions, one definite advantage would be that the spill of infected tuberculous material would not be emptied directly into the opposite dependent lung. The large bronchial passages would allow this spill to enter the trachea and with the head lowered slightly the material which had not been removed before operation would drain into the nasopharynx where if necessary it could readily be aspirated. In cases of pulmonary abscess where there was a large amount of pus present, this position has permitted the pus to drain freely into the gas mask instead of into the opposite lung.

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†Received for publication, August 26, 1933.

Several patients who were suffering from other than respiratory disease were selected, and the vital capacity was recorded when lying prone, supine, and on each side. In each instance it was noted that the most ideal position was the supine, since a slight decrease in vital capacity occurred in the others. If this is true, little doubt can exist as to choice of position. Certainly the tuberculous individual, all other factors being equal, would suffer the greatest decrease in vital capacity when the good lung was limited in its activity due to the patient's lying on the unaffected side. All of the tuberculous patients studied suffered a low-

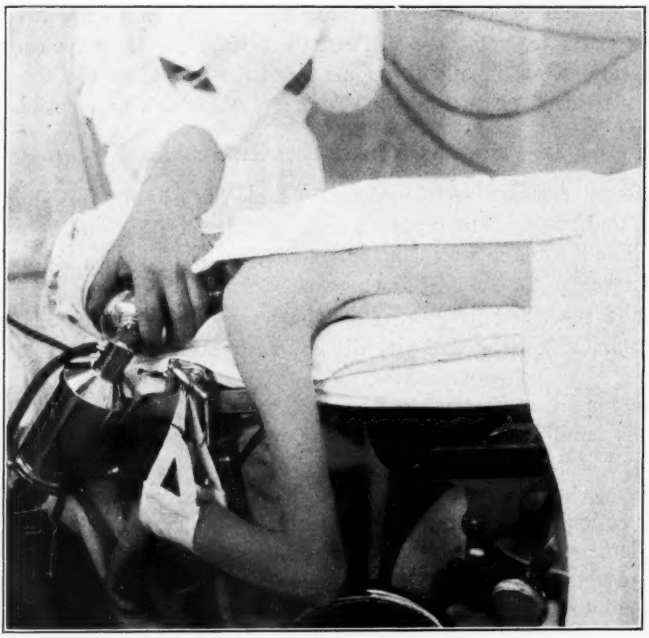

Fig. 1.—Patient in the prone position. The shoulder extends well beyond the edge of the table. The pillow beneath the shouder serves as a fulcrum and assists in displacing the scapula laterally when the arm is held by an assistant during the operation. The head is kept in a lower position than shown in the photograph.

ered vital capacity when lying on the unaffected side. This was to be expected in the presence of actual destruction of functioning pulmonary tissue. One serious objection to the prone position was mentioned; namely, that it would add to the difficulty of adequate exposure of the first rib at the first operative stage. This has been overcome by placing the shoulder on the side to be operated on about two inches beyond the edge of the table. The shoulder rests on a pillow. This serves two purposes: (1) the head does not have to be turned at an acute angle to accommodate the anesthetist's mask, and (2) respiration is easier

because the weight of the patient's body does not rest entirely upon the thorax. When the incision is carried well around the inferior angle of the scapula and the serratus magnus divided, no difficulty whatsoever is encountered. (Fig. 1.)

ANESTHESIA

Since postoperative pneumonia is one of the very serious complications of thoracoplasty, every effort should be made to choose a proper anesthetic method. The most ideal anesthetic for these cases must necessarily be one which is under second-to-second control and which insures an adequate amount of oxygen.

Local anesthesia has been extensively used. It possesses the advantage of not producing pulmonary irritation. Cough is under voluntary control. There are, however, certain disadvantages connected with its use. Not rarely novocaine poisoning has been reported. There is always the possibility of perforating a tuberculous pleura, and, in patients with low resistance, infection may develop more frequently through the infiltration necessary to obtain a satisfactory anesthesia. The ill-effects of such a prolonged procedure on particularly nervous patients cannot be denied.

Local anesthesia used in combination with ethylene or nitrous oxide is satisfactory. Less of both agents is required, and the objectionable features of local anesthesia alone are minimized.

In this country, nitrous oxide has been used extensively. When used alone it has the advantage of being the least unpleasant, during induction, of any type of inhalation anesthesia. It seldom stimulates cough if the induction is properly carried out. It can be kept very definitely under control from second to second, and recovery is rapid after the gas is withdrawn. This is advantageous in tuberculous cases in which early cough is considered necessary to empty the bronchial passages of infected material that has been spilled out of the cavity during the course of the operation. The following disadvantages of nitrous oxide may be encountered. If not carefully administered, during the induction violent cough will occur associated with hyperventilation and irregular jerking respiratory movements, all of which may result in the activation of latent tuberculous foci. Such danger can be readily overcome if the preanesthetic drugs are carefully judged and if care is taken to insure an adequate amount of oxygen supply during the induction. It may, in certain patients, fail to produce the desired anesthesia due to its limited anesthetic qualities.

Ethylene possesses the same advantages as nitrous oxide. Cabot and Davis[1] cite the added advantages of less sweating and heat loss during the administration. A 15 per cent deeper anesthetic level can be reached with ethylene than with nitrous oxide, still allowing the patient an adequate oxygen supply. Its chief disadvantage lies in its odor and ex-

plosiveness. Nitrous oxide-ethylene sequence is more desirable than ethylene alone, since the induction is more satisfactory to the patient.

Tribromethanol (avertin) has been used and has certain qualities superior to inhalation anesthetic agents. Induction is comparatively pleasant for the patient. The patient falls asleep without any of the troublesome respiratory difficulties often encountered in induction with an inhalation anesthetic. The blood pressure is markedly lowered unless the dose is accurately controlled and oxygen supplied. Bleeding is reduced to a minimum. The cough reflex is not abolished as is so often stated. It is not a safe anesthetic agent when used alone because the complete anesthetic dose closely approaches the toxic dose. In the patients in which we have used avertin alone a preliminary dose of morphine sulphate is given which is individualized and usually does not exceed gr. $\frac{1}{6}$ and scopolamine gr. $\frac{1}{150}$, one and one-half hours before operation. The observation of the reaction of these preliminary drugs aids in determining the amount of avertin to be used. The dose will range between a minimum of 65 mg. to a maximum of 120 mg. (average 90 to 100 mg.) per kilogram of body weight. The minute volume of respiration is decreased to such an extent that an oxygen rich atmosphere must be supplied. The patient may not waken for several hours after the operation, so immediate postoperative pain is reduced to a minimum. The combination of avertin and nitrous oxide provides a good anesthetic. The dose of avertin can be greatly reduced, thereby assuring complete safety, and the induction of the nitrous oxide is much easier. Where avertin has been used with a preanesthetic sedative of morphine and scopolamine, one can give nitrous oxide with an excess of oxygen, thereby minimizing the possibility of anoxemia. If the nitrous oxide is given alone, the oxygen concentration must be reduced and this adds to the difficulty of satisfactory induction.

Acetylene has been used most extensively at the Mayo Clinic. It has the advantage of permitting a high concentration of oxygen during its administration but has a disagreeable odor and is explosive.

Cyclopropane has been used at the Wisconsin General Hospital. We have employed it in four thoracic cases. It produces anesthesia with a very low concentration of the gas, only 20 per cent or less. It is irritating, however, and like ethylene and acetylene is explosive. Although promising, there has been insufficient clinical investigation to warrant its use. At present it is not available on the market.

In our experience the ideal anesthetic has been the nitrous oxide-ethylene sequence when there has also been properly individualized premedication with nonvolatile agents.

MECHANICAL

Certain well-known difficulties arise in the successful closure of cavities. Patients with thick walled cavities surrounded by a fibrous pleura

offer a definite mechanical problem. Most frequently these cavities lie in the apex but they may be centrally located. Extensive resections of long segments of rib by the posterior route may only succeed in pushing the cavity or cavities downward without effecting closure. This necessitates some other major procedure, and usually anterolateral thoracoplasty is the operation of choice.

Recently we have endeavored to eliminate the necessity of these additional major operations. Two minor adjunct procedures have been used; namely, preliminary parasternal chondrectomy which has been lately described and discussed by Alexander,[2] and the employment of pericostal sutures.

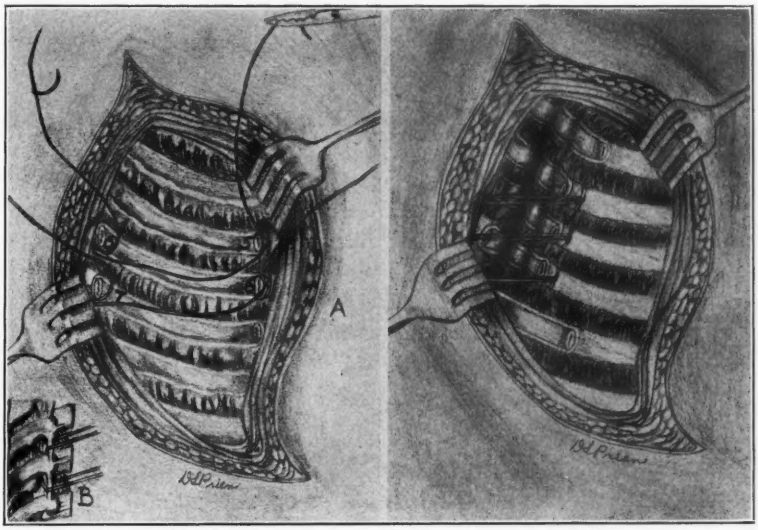

Fig. 2. Fig. 3.

Fig. 2.—*A*, A diagrammatic sketch showing the pericostal sutures tied around the posterior stumps of the anterior segments. *B*, Showing the intertransverse ligaments.

Fig. 3.—The sutures have been tied and the posterior stumps have been drawn close to the transverse processes. Note infolding of periosteum, intercostal bundles and pleura.

In patients in whom trouble is anticipated in securing the necessary collapse and where the operator finds the ends of the divided ribs forming a barrier to the closure of a cavity, the employment of pericostal sutures may solve the problem and eliminate an added major procedure or the reliance upon braces or pads to produce the collapse.

During the past two years, we have observed that by the placing of a suture around the posterior end of the anterior rib stump and then through the opposite intertransverse ligament, we were able to draw the cut segments close to the transverse processes. This produces a

maximum collapse with a moderate amount of resected rib. A suture of No. 2 chromic catgut is passed around the anterior segment about 2 cm. from the end. It is passed as close to the rib as possible, care being taken not to perforate the pleura. The suture is tied firmly about the rib, and then the needle is passed between the adjacent transverse processes one interspace below, thereby insuring two firm anchors. The assistant places his hand in the axilla and slowly bends the anterior segment backward so that its end is close to the transverse process. This produces not only an infolding of the parietal pleura, intercostal muscles, and periosteum into the gutter but reduces the diameter of the hemithorax as a unit. (Figs. 2 and 3.) When this method was first employed, it was feared that too much pressure could be exerted, especially on the left side, and that in certain instances the heart could be unduly displaced or compressed. This has not been found to be the case. In some cases it was mechanically impossible to bring the anterior stumps against the transverse processes because too much rib had been removed, but always a marked increase in collapse was obtained. We feel that it has been an extremely valuable procedure to patients who have a thin parietal pleura and manifest marked paradoxical breathing at the site of the resections. The anesthetist has checked this and noted prompt improvement in the patient's condition as soon as the sutures were tied. It is remarkable to see the diminution and almost complete elimination of the paradoxical movement. There is no doubt but that it is decreased in every case with the closure of the wound but certainly not as completely as when this method is employed.

The sutures are used only in the second and third stages. During the second stage the last rib resected at the first stage is always sutured along with those removed at the second stage. Due to the downward course of the ribs as they cross the axillary line the anterior segment is anchored to the transverse process of the adjacent lower rib.

Cavities in the central portion of the lung have also presented a problem of complete closure in certain instances when located on the right side. Since the diaphragm usually rises higher on the right following phrenicectomy, the liver may push high into the thoracic cage and act as a very effective tampon preventing adequate collapse at the base following rib resection. In centrally located cavities with this condition present, posterior resection is less efficient and collapse is more difficult to obtain. It has been possible, through the use of pericostal sutures to bring about a sufficient diminution in size of the thoracic cage just above the diaphragm, to close such cavities. (Figs. 4, 5, and 6.) In other instances the attempt has been made but unsuccessfully. The anterolateral approach can then be resorted to at an early or late date without any loss of time.

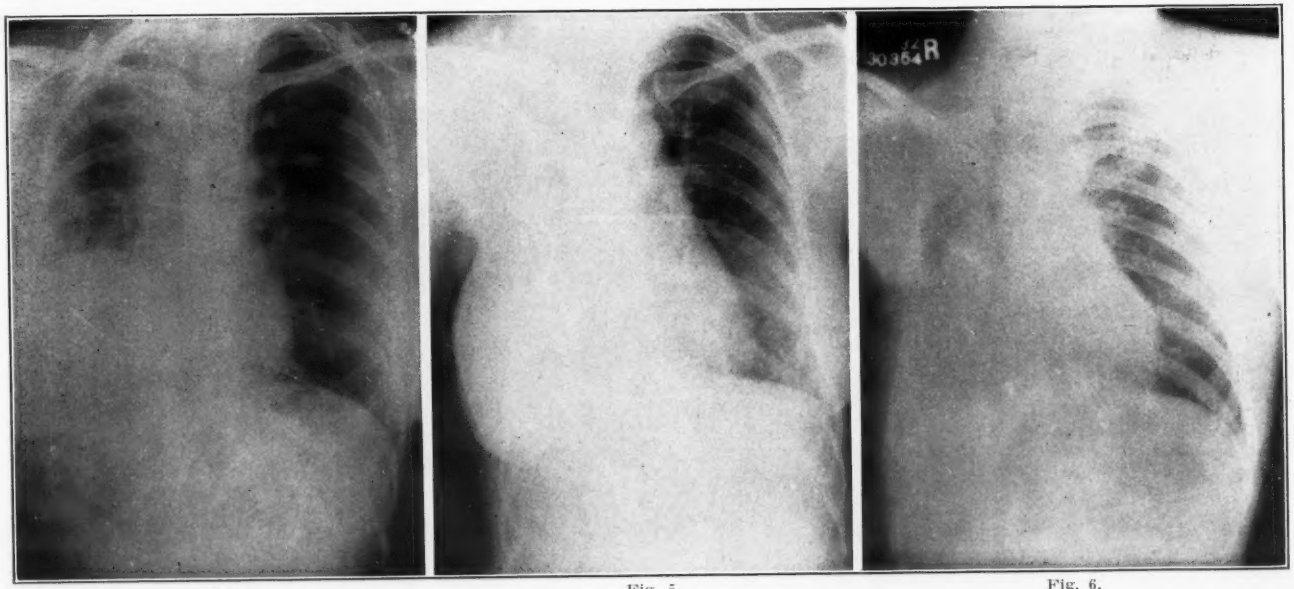

Fig. 4. Fig. 5. Fig. 6.

Fig. 4.—The x-ray picture shows patient with lesion on the right side. Phrenicectomy has been done followed by an excellent rise. The liver has also risen and forms a definite tampon.

Fig. 5.—X-ray picture of same patient as shown in Fig. 4 after three-stage thoracoplasty. With the aid of pericostal sutures excellent collapse has been obtained through forcing the liver downward at time of operation.

Fig. 6.—X-ray picture of patient following three-stage thoracoplasty with adequate amount of rib removed to produce collapse if pericostal sutures had been employed. Reoperation was successful in an adequate collapse.

POSTOPERATIVE CARE

The operative collapse of a tuberculous lung usually should comprise three or more major operations, after each of which the patient enters the most critical stage of treatment. The interval between stages must be long enough to allow sufficient convalescence from the previous stage and short enough to insure adequate collapse before the fixation of the chest wall occurs.

In order to accomplish this successfully, undivided attention must be focused upon the postoperative care. It is only in this way that the physical condition and morale of the patient can best be preserved.

Care should be taken to prevent chilling immediately after operation.

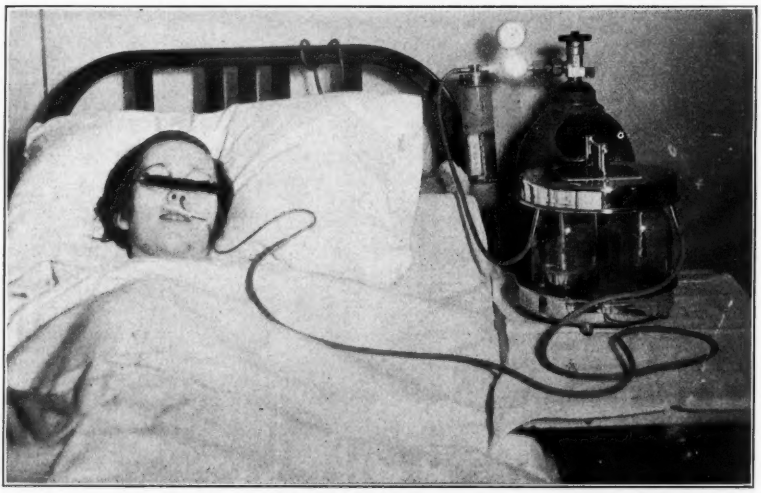

Fig. 7.—Patient receiving oxygen through intranasal catheter. The rate of flow is between four and six liters per minute.

This is overcome by moving the patient from the operating table directly into a warm bed where he is placed upon the operated side. At this time 1,000 c.c. normal saline are given intrarectally.

The blood pressure, temperature, and pulse are recorded at fifteen-minute intervals by the nurse. If the blood pressure is lowered, 500 c.c. of a 5 per cent solution of glucose are given intravenously over a period of not less than thirty minutes. If this is not effective, a blood transfusion is given. If the temperature falls below normal, artificial heat in the form of hot water bottles or an electric cradle is applied.

Just as soon as the patient has recovered sufficiently from the anesthetic and cooperation is possible, cough is demanded to insure against aspiration.

Before leaving the operating room the anesthetist inserts a No. 10 French catheter through the nares into the oral pharynx and fastens it so that the end will lie just above the tip of the uvula. Upon reaching the room, this is connected to an oxygen tank and oxygen saturated with water vapor is allowed to flow at the rate of four to six liters per minute. This has very definite advantages. It is much simpler than an oxygen tent and just as effective. It insures an adequate amount of oxygen at all times and prevents anoxemia which is so likely to occur immediately after any general anesthetic (Fig. 7).

Some patients before operation have a very low vital capacity, and doubt may exist as to whether they can successfully withstand the decrease necessarily brought about through the operative shock, toxemia, and diminution in functioning pulmonary tissue. During the first few days these patients need an adequate oxygen supply. It can be obtained only through an increase in the muscular effort of respiration. If this occurs over a period of time in patients with a low reserve, it may result in great fatigue and collapse. Through the use of oxygen therapy the oxygen concentration can be increased two to three times and the cardiorespiratory reserve maintained. Patients who ordinarily could not successfully undergo operation because of lowered vital capacity can be made safer operative risks.

In the usual case oxygen therapy is used during the first twenty-four hours. It is then gradually reduced until completely removed after thirty-six hours. If shortness of breath and increased pulse occur at any time while it is being decreased, the therapy should be continued. Care should be taken to cut the concentration slowly.

Pain should be controlled by morphine, and it is lessened if the operated side is supported during cough.

Fluids are given by mouth as soon as tolerated, followed by a rapidly increasing diet. The patient is usually ready for the next stage within two weeks.

Blood transfusion is routinely employed in tuberculous patients before operation and between the different stages in the presence of a secondary anemia. They are watched closely throughout their stay in the hospital, and transfusions are given when thought advisable. Transfusion has been found to be a valuable asset to improve the patient's general condition. The increased resistance produced by this therapeutic measure enables them to withstand the different operative stages with less reaction. Their convalescence following each stage is shortened.

WOUND INFECTION

Wound infection, although not a frequent occurrence, is a very serious complication. Any patient who has sufficient pulmonary involvement to merit thoracoplasty is much below par and has a lowered resistance to infection. The incision necessary to facilitate adequate exposure and

provide for the successful execution of the operation must be large. The trauma is more than that ordinarily seen in abdominal operations. With these conditions existing, it is imperative that every care be exerted to prevent its occurrence.

Next to pneumonia, infection has been considered one of the most severe complications arising during the operative collapse of a tuberculous lung. Unless infection is recognized early and adequate measures are instituted, the purpose of the operation may be defeated and the patient's life sacrificed. In a limited number of cases we have encountered pyogenic infection five times. Two occurred after the first stage and three after the second. In the first instance that infection was encountered it occurred after the second stage. We made the mistake of instituting conservative treatment in this instance and relied on the removal of a few stitches and the cigarette drain which was placed at the time of operation to take care of the infection. This resulted in an unnecessary delay until, when it was finally possible to perform the third stage, the periosteum had regenerated sufficiently to fix the thoracic wall and unsatisfactory collapse resulted. In the remaining cases more radical treatment was instituted. Just as soon as the infection was recognized, the incision was opened in its entirety and packed wide open, constant hot wet boric dressings were applied and Dakin's irrigations given every four hours. The result was good in each instance. Pain rapidly disappeared, and the temperature and pulse soon recovered their normal level. The infection cleared rapidly, and healing was so prompt that in three of the patients we were able to perform the following stage two weeks later through the open wound. In one patient, three weeks were allowed to elapse because the patient's general condition did not justify proceeding at the usual time. Infection did not develop in any of the successive stages even though healing was not complete in the granulating wound at the time of the next operation.

From limited experience, early adequate drainage is strongly recommended. This implies the removal of all sutures and the exposure of all recesses where infection may lurk. The scar following these radical measures has been satisfactory, and the final result of the operation has been good. No limitation of the shoulder has been observed.

SUMMARY

1. The clinical study of a limited group of cases indicates that the prone position possesses definite advantages when used in the performance of extrapleural paravertebral thoracoplasty.

2. In our experience the most desirable type of anesthetic agent at this time is the nitrous oxide-ethylene sequence.

3. Pericostal sutures employed during the second and third stages of a thoracoplasty are effective by reducing the transverse diameter of

the hemithorax as a unit and afford a method of preventing paradoxical breathing in patients with a very thin pleura.

4. The use of intrapharyngeal oxygen is recommended as a routine measure following thoracoplasty. It guards against anoxemia which if not treated will draw heavily upon the patient's reserve.

5. The use of blood transfusion before and between operative stages furnishes a very effective therapeutic measure.

6. Wound infection when present should be treated by early adequate drainage; otherwise the purpose of the operation may be defeated.

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SOME ASPECTS OF THE GROSS SURGICAL PATHOLOGY OF CHRONIC PULMONARY TUBERCULOSIS*

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THE purpose of this paper is to present some aspects of the gross surgical pathology of chronic pulmonary tuberculosis that seem of importance in the estimation of the indications, type, and results of collapsotherapy. One cannot hope to do more than delineate some observations that have helped to define a rationâle and predicate a mode of treatment. It is not the purpose of this paper to make a synopsis of the well-known literature, except so far as it has a bearing on those phases of the gross pathology of pulmonary tuberculosis that will be discussed.

In brief, the paper will deal chiefly with the correlation of bronchographic and operative observations to the pathogenesis and gross aspects of chronic pulmonary tuberculosis. The subject will be taken up in three sections: (a) the currently accepted concepts of the gross pathology of chronic aerogenous pulmonary tuberculosis and their relation to (b) the bronchographic observations in chronic tuberculosis and (c) the pathology as noted in the operation of pneumocavernolysis.

A. GROSS PATHOLOGY OF CHRONIC PULMONARY TUBERCULOSIS

The picture of chronic tuberculosis of the lungs is so variable that it may well be said that no two tuberculous lungs are alike. The protean gross aspects of the disease may be described in a general fashion along the following lines.

There is practically unanimous agreement that in aerogenous tuberculosis the site of onset of the lesion is in the third to fifth order bronchi, in the fine interlobar bronchioles and end bronchi.[2] The initial lesion begins within the bronchioles, first in the mucosa and then spreads to the infundibuli and alveoli with the formation of a tuberculous lobular pneumonia. The fate of the first foci varies; fibrous proliferation encapsulates and heals them, or they become starting points of various forms of tuberculosis which extend contiguously, or after remaining stationary for a time, become progressively active again with hematogenous, lymphatic, and intracanalicular dissemination.[1, 3, 4, 5]

The alveolar parenchyma and the bronchi may participate concur-

*Read by Title at the Sixteenth Annual Meeting of the American Association for Thoracic Surgery, Washington, D. C., May 9, 10, 11, 1933.

rently and inflammatory exudation may play the prominent part as in tuberculous pneumonia. This may entirely resolve. The caseous products may quickly liquefy, be distributed by way of the bronchi and thus infect larger contiguous or distant areas. In other cases the alveolar parenchyma may remain comparatively free of disease and the process show a tendency to spread along peribronchial and perivascular lymphatics with the formation of nodules in the interstitial tissue. Or caseous bronchial and peribronchial lesions are combined and stimulate exudative processes in their own and adjacent parenchymal territories. The lesion is further modified by softening of the caseous masses and cavity formation, which changes may, however, remain entirely absent in others. Indurative healing processes due to reactive connective tissue proliferation develop in the vicinity of the tuberculous process in some lungs; in others, none occurs. If very active connective tissue proliferation sets in, the progressive changes may be checked and many foci of smaller and even larger extent may undergo cicatricial healing.[1, 3, 4, 5]

It cannot be the aim of this paper to assign the rôle of tuberculous bronchitis in the pathogenesis of the disease. Suffice it to say that bronchial lesions are commonly found in contiguity to the tuberculous foci.[1, 4, 5] Although there is practically a unanimity of opinion that the initial lesion is bronchopneumonic, the widespread area of tuberculous bronchial involvement as commonly found in the proximal vicinity of the lesion may be the result of bronchial infection by tuberculous discharge issuing from the more peripherally situated cavity.[2]

It is of importance in this connection to describe briefly the bronchial lesions in order to correlate the bronchographic aspect of the disease to be described later. The most common sites for the first bronchopneumonic lesions are the posterior apical and the paravertebral segments of the upper lobes.[2] The apical and paravertebral bronchi are therefore the ones that are usually most extensively involved. At first an annular tuberculous infiltration may narrow or occlude the bronchial lumen with collapse of lung tissue. Disintegration of the infiltrate leads to patency of the lumen. Multiple cicatricial stenoses, a tuberculous annular stenosing infiltration, and bronchial ulcers have been observed. Very commonly there is a simple ulcerative or a caseous bronchiectasis.[1] Ulcerating bronchiectatic cavities with or without caseation are frequent. It is not always easy to differentiate between a bronchiectatic ulcerating cavity and an old smooth lined ordinary cavity such as may be found in softened caseous lung tissue.[5]

Cavitation, the hall mark of pulmonary tuberculosis and the objective of collapsotherapy, therefore has a definite relation to the bronchopneumonic segmental localization that has been emphasized. Tuberculous cavities may arise on the basis of (a) liquefaction and sequestration in caseous bronchopneumonia areas, (b) endobronchial

and peribronchial caseation and disintegration, and (c) tuberculous ulcerative bronchiectasis.[1] The size and form of early cavities depend entirely on the caseous focus from which they develop. If they arise from a bronchopneumonic focus, the cavity is of course very small at first and more or less rounded or circumscribed; if they appear in a caseous pneumonic area of lobular or lobar dimensions, then multiple liquefying foci are present which by their confluence form extensive irregular systems of cavities. It may again be mentioned that the continuous discharge from cavities exposes the contiguous larger bronchi in the local lung sector to tuberculous infection. Cavitation arising in a caseating bronchopneumonic area is usually peripherally situated, having but a rim of compressed tuberculous alveoli between it and the pleura. Smaller areas of necrosis may, however, occur in more central areas of caseation. By and large, chronic cavities of any moment are subpleural.[1, 5]

To recapitulate, emphasis has been placed on the initial bronchopneumonic lesion in aerogenous pulmonary tuberculosis, its evolution within specific bronchopulmonary segments, and the frequency with which the bronchi of those segments are grossly involved.

B. BRONCHOGRAPHY

It was therefore thought that bronchographic amplification of the roentgen film would be of value in indicating the location of cavitation for collapsotherapy through the revealed relationship of the cavity to the known anatomy of the radio-opaque bronchial tree.

The literature on bronchography in pulmonary tuberculosis has indicated the following: (1) Cavities may or may not be entered.[6-9, 10, 18, 20, 25] (2) Bronchiectasis contiguous to the lesion and basal bronchiectasis are common.[12-17, 19-22, 24] (3) Bronchiectasis within the zone of the tuberculosis has been seen in every stage of the disease from the early pneumonic infiltrate to fibroid phthisis.[11, 15, 18, 22-25] (4) Postoperative bronchography may be of value in disclosing a basilar bronchiectasis as the causal factor for copious postoperative expectoration.[12, 13]

TABLE I

LESION	MIXED CAVERNOUS	FIBRO-CAVERNOUS	CASEO-CAVERNOUS	INFILTRATION	HEALED CAVITIES	TOTAL
Bronchial Dilatation	27 – 73%	9 – 60%	1 – 25%	3 – 43%	0 – 0	40 – 61.5%
No Dilatation	8 – 21.6%	3 – 20%	3 – 75%	4 – 57%	1 – 50%	19 – 29.3%
Suspicion of Dilatation	1 – 2.7%	0 – 0	0 – 0	0 – 0	1 – 50%	2 – 3.1%
Distortion of Bronchi	1 – 2.7%	3 – 20%	0 – 0	0 – 0	0 – 0	4 – 6.2%
Total	37	15	4	7	2	65

At the Hudson County Tuberculosis Hospital and Sanatorium 95 lipiodol bronchographies have been done (intratracheal catheter technic) without the occurrence of major complications. Three instances of transient febrile reactions and one of transient angioneurotic edema have occurred. Sixty bronchograms, as closely studied, yield the information[26] shown in Table I.

Of the 65 cases, 56 were cavernous lesions of one type or another. Of these, 66 per cent showed definite bronchial dilatation; 7 per cent bronchial distortion without dilatation, and 2 per cent a suspicion of dilatation. In this series mixed cavernous lesions showed the highest incidence of bronchial dilatation, 73 per cent; fibrocavernous, 60 per cent; and caseocavernous, 25 per cent.

Infiltration showed the remarkably high incidence of 43 per cent bronchial dilatation. The infiltrated lesions showing this dilatation were all predominantly productive.

Six and two-tenths per cent of cases showed distortion of bronchi with no attending dilatation. All were associated with changes in lung volume and were probably due to fibrosis.

In 80 per cent of the 40 cases showing bronchiectasis the dilatation was confined to the upper lobe, and in the remaining 20 per cent, two or more lobes were involved. In 25 per cent of the cases with cavitation, entry into the cavity was made with the demonstration of a fluid level.

Our bronchographic observations reveal bronchiectasis in tuberculosis to exist in two anatomically different forms. Contiguous to the upper lobe cavernous lesion, the ectasis is usually confined to the lobular distribution of the bronchopneumonic lesion, although if the lesion is large all the lobar bronchi may be involved. The most common involvement, as to be noted more fully later, is in the postapical and paravertebral branches. Such bronchiectasis may be tubular and not uncommonly is beaded, grape-like, and sacculated.

In contrast to this is basilar bronchiectasis which not infrequently occurs, most notably in fibrocavernous disease. It most commonly is seen at the base of a contracted hemithorax containing a large upper lobe fibrocavernous lesion and is usually associated with marked evidences of fibrosis and mediastinal displacement to the affected side. It should be stated, however, that no absolute association of atelectatic fibrosis and basilar bronchiectasis exists. Contracted hemithoraces have occasionally been seen with bronchograms of the base normal apart from distortion, and conversely, marked dilated basilar bronchi have been noted without evidences of fibrosis or contraction of the affected hemithorax. The basilar bronchiectases usually are lobar, markedly dilated, often bizarrely giant-sized, and have a "glove fingers" outline. Their appearance is quite unlike the bronchiectasis seen in the region of the upper lobe disease.

Our observations on bronchography may be classed under two heads: (a) localization and (b) diagnosis.

Localization.—For purposes of selective surgical collapsotherapy, more specifically with reference to pneumocavernolysis, the precise location of the cavernous lesion with respect to the chest wall is of great importance. Inasmuch as aerogenous pulmonary tuberculosis begins as a segmental lesion, as a bronchopneumonia, and is associated with demonstrable contiguous bronchiectasis; bronchography, through its demonstration of the topography of the associated bronchi and bronchial lesions and the anatomic relation to these to the cavernous infiltrate, affords a distinct localizing aid. The cavity itself has not often been penetrated. Nevertheless a demonstration of the bronchial tree in relation to the cavity has obviously equal significance in localization of the cavity.

The relationship of the radio-opaque bronchial tree to the chest wall enables one to localize more or less exactly the parietal representation of the cavitation for purposes of operative attack. The value of this in selective operative collapsotherapy needs no further emphasis.

In upper lobe lesions the bronchi, most commonly the site of bronchiectasis contiguous and leading to the cavitation, have been the posterior apical and paravertebral branches of the upper lobe bronchus. The bronchiectases have been tubular and sacculated and with good filling the specific branches are readily defined. The bronchi are commonly rather widely dilated, bizarrely deformed and usually folded up in a fanlike manner. Not uncommonly with large lesions all the lobar branches have been involved. However, it is to be noted that bronchiectasis although common is not constant, cases of upper lobe cavitation having been seen in which contiguous bronchiectasis has not been present.

Diagnosis.—Bronchography has served to amplify and greatly extend the significance of x-ray examination. In effect, it enables one to explore and grasp details of the gross surgical pathology that would otherwise completely escape detection.

One may anatomically group the various diagnostic uses of bronchography into (1) bronchial, (2) parenchymal, (3) pleural.

1. *Bronchial.*—Not uncommonly in upper lobe cavernous lesions bronchography demonstrates that the involved area is the entire lobe. The entire bronchial tree of the lobe is visualized as a group of folded-up, fan-like, ectatic bronchi within a greatly shrunken upper lobe bounded inferiorly by the sharply curved interlobar fissure. It becomes evident that the lesion is a fibrotic shrunken upper lobe containing a cavity. The significance of such a disclosure is apparent when consideration is taken of the futility of employing a phrenicectomy, for example, in order to affect such a lesion. From the appearance of a contracted

shrunken lobe containing closely neighboring ectatic bronchi an inference of the chronicity of the lesion is warrantably drawn. Bronchography thus serves to depict the amount of parenchyma between the bronchial arborizations, and by this revelation of contiguously packed deformed ectatic bronchi, with little intervening parenchyma, leads to a warrantable assumption of chronicity and fibrosis of the lesion. It has been found that the history of a case and the ordinary x-ray examination are not as important clues to the chronicity and fibrosis of a lesion as the bronchographic appearance. The confirmation of this has been seen in the operative pathologic findings. Conversely, the demonstration of a lobar bronchial tree of apparently normal size and extent, with or without striking bronchial changes contiguous to the cavernous lesion, suggests that minimal fibrosis exists.

2. *Parenchymal.*—Circular areas in the vicinity of cavities, commonly referred to as "moth-eaten" or "honeycombing" are clearly defined by bronchography in most instances as being sacculated bronchiectases contiguous to the cavity. Such information is of great importance in estimating the necessary degree and extent of surgical collapsotherapy.

In cases of unilateral total opacity in which all structural detail is lost, bronchography serves to outline the bronchial tree and parenchyma and thus determine the presence and site of bronchiectasis, pleural effusion, atelectasis, fibrosis, etc.

Areas of translucency suspicious of cavitation may be accurately defined as being either artefact, bronchiectatic, or cavities. The ectasis usually is well depicted, whereas entry into cavitation is made but rarely and is then visible as a thin fluid level.

3. *Pleural.*—By outline of the limits of pulmonary parenchyma the extent and type of pleural involvement may be defined, be it caused by tissue, fluid, or air. Effusions and sacculated pneumothoraces have been recognized as such only by means of the bronchogram.

Annular shadows, thought in some quarters to be due to "pleural rings," have, in several instances, been entered bronchographically, demonstrating their cavernous nature.

A special reference is to be made to postoperative bronchography. The interpretation of films taken after chest collapsing operations is of the greatest importance in the prognostic estimation of the results of surgery and in the indications for further operative effort. Unfortunately, however, the postoperative deformity and shadowing render reading of the films extremely difficult. Areas of translucency residual after operation need further definition. Bronchography has been of considerable aid. On some occasions it has demonstrated bronchiectasis as the reason for areas of rarefaction or conversely, the iodized oil has entered and filled a residual cavitation which has resisted the first surgical attack.

Copious nonbacillary sputum may be accounted for by bronchography as due to the contiguous and basilar giant bronchiectases.

C. GROSS OPERATIVE PATHOLOGY

The gross pathologic findings to be described are those encountered in the operative procedure of pneumocavernolysis as done by Dr. Harold Neuhof for chronic cavernous pulmonary tuberculosis. An essential feature of the operation is localization of the cavernous lesion with the purpose that selective direct cavity-collapsing therapy may be effected. With the knowledge at hand of the parietal representation of the pulmonary segments as revealed by bronchography, a fairly precise localization of the cavity on the chest wall can often be secured.

The operative procedure has for its initial goal the visualization and palpation of the subpleural aspects of the cavernous lesion in order that collapsing compression can be directly induced. The gross aspects of the subpleural cavernous lesion as it appears at operation will now be described.

The cavity as located by bronchography and corroborated by operation usually lies in the area of the paravertebral and postapical bronchus distribution, namely, posteriorly and somewhat posterolaterally. The lesion is not uncommonly found at some distance inferior to the arc of the first rib. When operation has disclosed the lesion, it has always been found to be subpleural.

After removal of lengths of such ribs as is indicated by x-ray and bronchography, varying immediate degrees of depression and respiratory movement of the decostalized operative field are to be observed. In general, it may be said that the more pronounced the depression and the respiratory excursions, the less likely is one over the site of cavitation. However, minimal fibrosis about a cavity may permit considerable depression and respiratory movement after rib removal. Not uncommonly after decostalization the peripleuritic tissues and pleura remain practically unchanged, standing up as an unaltered rigid shell. In such cases there is a marked peripleuritic scarring fibrosis and induration which is observable and palpable. It is this type of lesion which indicates the futility of decostalizing operations as the sole procedure for collapse of such cavities. Effacement of this type of stiff cavity requires direct lysis to free the cavity from its parietal adherence.

After decostalization direct inspection and palpation by findings of peripleuritis, immobility, induration, firmness, resistance, and infiltration usually give the lead to the site and extent of the lesion. After severance and reflection of the intercostal bridges and periosteum the pathologic process is studied. Adhesions sealing off the free pleural cavity over the cavernous lesion have invariably been present. Ad-

hesions may be thin and filmy over the less fibrotic lesions or dense, scar-like, and deforming over the old stiff-walled cavities.

The surface of the cavernous lesion, when seen after reflection of the adherent thickened, occasionally unidentifiable, layers of tissue binding it to the parietes, is seen to be of varying size, discolored greyish-white with areas of anthracotic pigmentation. Distinct scarring with puckering of its surface is not infrequently noted. The following appearances have thus far been observed:

(a) The surface may be seen to be indented in a cup-like manner by the immediate action of the atmospheric pressure and the retractile recoil of the lung upon the released lesion. It is then identified as a softly fluctuant area, having a distinctly demarcated circular rim of infiltration and resistance.

(b) Scarring over the site of cavitation may be such as not to permit complete effacement and the leathery tough visceral pleuritic membrane will then be seen to be puckered and wrinkled. Such a plaque of scar tissue may extend over a large area contiguous to the cavitation.

(c) The cavity may appear as a soft boggy swelling, appearing to fill with inspiration and collapse somewhat with expiration with an apparent air content, which is displaceable easily within the swelling and is readily reducible by gentle manual manipulation. Upon one occasion such a swelling collapsed completely without manipulation, directly under visualization, with the audible accompaniment of a hissing sound.

(d) The lung (usually anterior and caudad to the upper lobe lesion) that is free from gross disease and hence not requiring collapsotherapy, is identified by its pink color, crepitant air content, absence of resistance and infiltration, and free mobility with respiratory excursions.

Second stage operations for cavities residual after pneumocavernolysis reveal several pathologic findings meriting comment. The pleura beneath the iodoform packing, which has been introduced to maintain compression, is quite enormously thickened (approximately ¼ to ½ inch thick) consisting of a solid lamella of dense, tough, fibrous tissue throughout the area subjacent to the pack. This membrane is thick and firm enough to restrain the lung beneath it from respiratory movement and appears as a rigid shell.

The "dead space" formed by the compression and retractility of the lung is found to be a variously sized pocket, lined by fibrous immobile tissue and often containing a small amount of clear serous fluid.

SUMMARY

1. A brief review of the pathology of chronic aerogenous pulmonary tuberculosis has been made, stressing the bronchopneumonic pathogenesis.

2. Important aspects of the gross surgical pathology of chronic pulmonary tuberculosis may be illustrated by bronchography.

3. Through bronchography one is enabled to arrive more nearly at an estimation of the exact location and type of the lesion under consideration for operative treatment.

4. Some gross surgical appearances of tuberculous cavernous lesions as seen at selective operative treatment for cavitation, namely, pneumocavernolysis, have been described.

I wish to express my very great appreciation and indebtedness to Dr. Harold Neuhof, thoracic surgeon of the Hudson County Tuberculosis Hospital and Sanatorium, for permission to have observed and studied this material from his service, and to thank Dr. B. S. Pollak, director of the Hudson County Tuberculosis Hospital and Sanatorium, and Drs. B. P. Potter and J. E. Murphy of that institution for their courtesy and cooperation.

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EMPYEMA IN PULMONARY TUBERCULOSIS*

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IN THE hope of yielding some light on the problem involved in the management of empyema in the course of pulmonary tuberculosis, all instances of such effusions which have occurred at the Olive View Sanatorium in the period of 1921 to 1931 have been carefully investigated and the observations analyzed for possible significance. It is noteworthy that during the first years of this period, little, if any, treatment was accorded in such cases, and when it was, resulted in insufficient surgery and, consequently, insufficient drainage; while in the latter part of the period there was an increasing evidence of more active and vigorous treatment, with more extensive surgery and with better drainage established and, consequently, better results.

During this decade, there were more than 6,000 admissions to this institution, including more than 4,500 patients suffering from pulmonary tuberculosis, about three-fourths of whom were in the advanced stage of the disease at the time of admission, cavities occurring in 80 per cent of the total number. Among these patients, there have been 250 cases of pleural effusions, exclusive of a large number in which evidence of a limited amount of fluid could be seen in the x-ray films, following induction of artificial pneumothorax. Of these patients, constituting more than 5 per cent of the 4,500 patients suffering from tuberculosis, 71, or 1.6 per cent, were found, on examination, to suffer from a real empyema; that is, to have a purulent or thick exudate in the pleural cavity, while the remainder, constituting about 4 per cent of the entire number of cases, were rather of a serous and seropurulent character.

Further examinations showed that of the purulent cases of empyema, organisms other than the tubercle bacillus could be detected in but 23; which were grouped separately under the heading of mixed infection or pyogenic empyemas.

We shall consider, therefore, three different groups of cases:

First: Cases with mixed infection purulent, in which there were, in addition to the acid-fast organisms, other germs, mainly staphylococci or streptococci, presenting a picture of sepsis with high fever and leucocytosis, and rendering the prognosis more serious. The pleural exudate, when withdrawn, was usually thick and opaque, and contained many leucocytes and many pyogenic microorganisms, either

*Received for publication February 12, 1934.
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on stained smears or on cultures. Sixteen had acid-fast bacilli in the pleural fluid; two were negative, five not examined, although the latter were far advanced pulmonary patients having positive sputum; some were terminal cases with open sinuses, living but a few days after admittance to the sanatorium.

Second: Cases of simple tuberculous empyema, in which purulent exudate was obtained but examination revealed only the tubercle bacilli. There is not, of course, a hard and fast line between the purulent and seropurulent fluids, but those described as thick or filled with pus were listed among the purulent cases; while those in which the exudate although turbid or cloudy was still readily liquid, and those in which the fluid was never completely examined (including a few mild dubious cases) were listed seropurulent and classed with the serous cases.

Third: Serous cases or those with clear or slightly cloudy fluid.

The general prognosis as regards the effusions per se as shown in Table I varies considerably in these three types of effusions. By "cured" we mean empyema eradicated and underlying tuberculosis of the lungs arrested. "Clinically well" means eradication of the empyema. "Improved" means the patient is still in the hospital but apparently better of empyema or awaiting further surgery. In 23 cases in the mixed group, 52 per cent, or more than half, improved and 43 per cent died as the result of the effusion. In 48 cases in simple tuberculous empyemas, 51 per cent improved, but these were by no means an indifferent syndrome, as here also the mortality rate was 42 per cent in 48 effusions; strongly contrasting to this, were 179 cases of serous or seropurulent effusions, in which group less than 20 per cent succumbed and 61 per cent were improved. Here, I wish to call attention to an important factor, namely, the difference brought out in Table V and Table VI, the comparison of the results of the cases treated surgically and those receiving no surgical treatment.

TABLE I

TYPES OF TUBERCULOUS EFFUSIONS AND END-RESULTS

	TOTAL	CURED	CLINICALLY WELL	IMPROVED	STATIONARY	WORSE	DEAD
Mixed infection	23-10%	43%	10%		4%		43%
Purulent tuberculosis	48-19%	31%	20%		5%	2%	42%
Serous and seropurulent	179-71%	33%	14%	14%	12%	8%	19%
Total	250	32.8%	14.8%	9%	10.4%	5.3%	27.7%

There were a few more women than men in these series, probably reflecting the fact that there are somewhat more women than men in the entire institution, also perhaps because of the greater tendency

of the physicians on the women's side to resort to the use of artificial pneumothorax, which is one of the predisposing factors in the production of such effusions. This is emphasized by the fact that the men had more of both types of purulent effusions. There was a relatively better prognosis of empyema in the women as compared to that in the men, the women showing the lower mortality rate, particularly among the mixed infection group. Altogether, in this series only 27 women and 39 men died, although there were actually more women than men in the entire series. Again, it is well to note that artificial pneumothorax as a predisposing cause, increased the incidence of empyema, and also is a factor in the lower mortality rate.

The age incidence in the patients varied from a few children below their teens to several elderly patients of threescore years or more. The average age, however, was between twenty and thirty years—nearly one-half of the patients coming in that decade. This was true for each of the three types of effusions. The prognosis appears to be slightly better, also, in this group of the twenties, both younger and older patients showing a slightly higher mortality in all three types of classifications.

The side of the chest involved was more often the left side, 132 in all, while 5 were bilateral cases all of which occurred in the serous or seropurulent type. The cases of left side involvement appeared to have a better prognosis than those in which the exudate occurred on the right side, which is, in accordance with the more generally known facts in the case of artificial pneumothorax and probably is related to the anatomical structure of the chest and the greater ratio of left-sided artificial pneumothorax cases, with an associated thickened pleura which probably presents a barrier to the ready absorption of toxins.

TABLE II

STAGE

	MIXED INFECTION PURULENT				SIMPLE TUBERCULOSIS PURULENT				SEROUS AND SERO-PURULENT				TOTAL
	IMP.	WORSE	DIED	TOTAL	IMP.	WORSE	DIED	TOTAL	IMP.	WORSE	DIED	TOTAL	
Min.					3		2	5	10		4	14	19
Mod.					1			1	18	1	2	21	22
F. A.	12	1	10	23	22	1	19	42	104	11	29	144	209
Total	12	1	10	23	26	1	21	48	132	12	35	179	250

More than 80 per cent of these patients were cases of far advanced pulmonary tuberculosis at the time of admission, and it is in this group that the prognosis was, as may be expected, poorest. The results, however, in the small number of instances in the minimal cases was much graver than was expected, suggesting that in these cases

the effusion was a matter of particularly grave significance, the pleura in these cases being less thickened, and the absorption of toxins possibly facilitated.

More than 80 per cent of these patients showed acid-fast bacilli in the sputum, and it was in this group, as might be expected, that the mortality was highest—91 per cent of the total number of deaths. The patients with negative sputum represented generally lighter forms of the disease. In some instances there was no expectoration, possibly because of compression of the diseased lung by fluid.

The exudate from the chest was sent to the laboratory in 120 instances, and in 60 per cent of these it was found to contain acid-fast bacilli. Undoubtedly, this figure would have been much higher if larger samples of fluid had been sent for examination, or if a method of concentration, culturing or animal inoculation had been more frequently employed in the earlier periods. As might have been expected, the prognosis was somewhat better in those instances in which organisms were not found, since they may have represented lighter degrees of the pleural infection, or merely allergic or mechanical affections.

TABLE III

PLEURAL FLUIDS

	MIXED INFECTION PURULENT				SIMPLE TUBERCULOSIS PURULENT				SEROUS AND SERO-PURULENT				TOTALS
	IMP.	WORSE	DIED	TOTAL	IMP.	WORSE	DIED	TOTAL	IMP.	WORSE	DIED	TOTAL	
Pos.	9	1	6	16	17	1	11	29	20	1	8	29	74
Neg.			2	2	6		1	7	29	1	7	37	46
Not Ex.	3		2	5	3		9	12	83	10	20	113	130
Totals	12	1	10	23	26	1	21	48	132	12	35	179	250

In Table III, 46 patients whose pleural fluid yielded no tubercle bacilli were all cases of far advanced pulmonary tuberculosis, except three, who were moderately advanced. Only 6 of these 46 had negative sputum as well as negative pleural fluid. One of the six had a bronchial fistula, while another's history was prior to residence at Olive View. It is felt this leaves little doubt as to the character of the fluid and the presence of a tuberculous empyema.

The chief etiologic factor in the mixed infection group of cases apparently was manifested prior to admission to this institution, since 7 of these 23 patients entered the institution with chests already infected and lived an average of fifty-two days. In the remaining cases of this group and the other groups, however, the employment of artificial pneumothorax was, undoubtedly, a chief agent to be held responsible. Thus, of 500 patients treated with artificial pneumothorax in this institution, slightly less than 23 per cent developed

TABLE IV
EMPYEMA ETIOLOGY

TOTAL	CURED	IMP.	WORSE	DEAD	CURED	IMP.	WORSE	DEAD	TOTAL	CURED	IMP.	WORSE	DEAD	TOTAL DEAD (%)
Artificial Pneumothorax Before (Male)														
42				3	4	5		2	11	6	16		6	11.8%
(Female)														
77	6	2			8			8	16	16	22	6	9	14.2%
Spontaneous Pneumothorax Before														
26	2			3		5	1	8	14		3	2	2	50.0%
Pleurisy														
60				2				1	1	26	11	4	16	32.0%
Pneumolysis														
3						1			1	2				
Unknown														
42	2	1		2	2	1		2	5	8	22		2	14.5%
Total														
250														

pleural effusion sufficiently large, or grave enough, to be clinically recognized; while, of the other 4,000 patients, not so treated, effusion followed spontaneous pneumothorax in 26 instances, and simple pleuritis in 60 others, a total incidence of 3.2 per cent.

The highest mortality is associated with a group following spontaneous pneumothorax, 50 per cent; the next highest mortality is that following pleurisy, 32 per cent; and the least 26 per cent, following artificial pneumothorax. The danger of contamination of the pleural cavity by way of the lung is, therefore, of less serious consequence when following artificial pneumothorax than either from pleurisy or spontaneous pneumothorax, and this is emphasized further following more active treatment accorded empyema patients.

Prior to the formation of the pleural effusions or empyema of the cured and improved in the mixed and pyogenic type, 48 per cent had had pneumothorax; in the purulent type, the same was true in 56 per cent of the cured and improved; and of the cured and improved in the serous and seropurulent 45 per cent had pneumothorax prior thereto.

Complications were dealt with in 25 instances—the most frequent serious complication being bronchial fistula, while associated conditions included tuberculosis in other systems of the body, in addition to the pulmonary tuberculosis, and particularly peritonitis.

Separating all cases into two groups: First, we have the untreated surgically; second, the surgically treated, which includes aspiration and aspiration with some chemical, continuous open and closed drainage, phrenicoexairesis, costectomy, thoracoplasty, modified Schede operations and combinations of these. One hundred and twenty-eight, or 51.2 per cent, of all the cases fell into the first untreated group. The course and results were such as to indicate that this was a matter of culpable neglect, since the mortality was considerably higher than in the surgically treated group. The efficacy of treatments varied, however, in the different surgical procedures and in the different types of empyemas. In the mixed infection with tuberculosis, the prognosis was slightly better than in the pure tuberculosis empyema.

One patient of the mixed type was not treated and died. Of the 22 surgically treated in this same group 40.8 per cent, or 9 died; while 43.5 per cent were cured and 13.7 per cent improved. Of this same group, 9 were treated by aspiration or aspiration and some chemical, the result being that 2 died, 2 improved, 5 (55.5 per cent) were cured. This shows somewhat better results and suggests that aspiration or aspiration with some chemical instilled is the first choice and, if unsuccessful, should be followed by thoracoplasty in conjunction with aspiration and, if necessary, costectomy and ample drainage or modified Schede operation.

TABLE V

TUBERCULOUS EFFUSIONS

	MIXED WITH PYOGENIC				PURULENT				SEROUS AND SEROPURULENT					TOTAL
	CURED	IMP.	WORSE	DEAD	CURED	IMP.	WORSE	DEAD	TOTAL	CURED	IMP.	WORSE	DEAD	
Not treated				1	1	1		9	11	32	48	9	27	128
Aspiration	2	2		1	2	6		7	15	13	14		5	52
Aspiration with chemical	3			1	4	3	1	3	11	3	5	2		25
Drainage closed											2	1	1	4
Drainage open				1				2	2					3
Aspiration phrenic					1				1	4			2	7
Phrenic										4	5			9
Phrenic open drain										1				1
Phrenic thoracoplasty	1				2				2	1				4
Thoracoplasty	3			1	1				3					7
Thoracotomy open drain	1	1		5	2				2					9
Pneumophrenic thoracoplasty					1									1
Total	10	3		10	14	12	1	21	48	58	74	12	35	250
Pneumo prior to	6	2		3	12	5		10		22	38	6	15	119

Failures in a few of these cases were due, doubtless, to lack of enough drainage and collapse of the chest wall to reduce sufficiently the cavity or secure apposition of the pleural surfaces. In the purely tuberculous empyema cases, 12 or 32.4 per cent that were treated surgically died, 35.7 per cent were cured, 29.7 per cent improved and 2.8 per cent became worse. In the seropurulent 12.6 per cent of those surgically treated died, 41.2 per cent improved, and five became worse. Of all types treated surgically, 40.1 per cent were cured, 32.7 per cent improved, 3.5 per cent became worse and 23.7 per cent died.

Table V shows the different methods of treatment, the number of cases of each or combinations of same. Two of these methods need further explanation. In these cases, open drainage was attempted by insertion of a small rubber tube through a cannula and leaving it with suction. Open drainage, on the other hand, made use of a larger tube which was left open with a gauze dressing. A trocar cannula the size of a No. 32 French Sound is now used, with rubber tube to fit for closed drainage where aspiration cannot be carried out. This now frequently precedes costectomy, and open drainage.

TABLE VI

ECONOMIC STATUS AND TREATMENT

	TOTAL	WORKING	LIVING	LOST	DEAD
Surgically not treated	128	17 or 13.2%	25 or 19.5%	21 or 16.4%	65 or 50 9%
Surgically treated	122	15 or 12.2%	44 or 36.0%	3 or 2.5%	60 or 49.5%
Total	250	32 or 13.0%	69 or 27.0%	24 or 9.6%	125 or 50.4%

TABLE VII

LONGEVITY AND PRESENT ECONOMIC STATUS PER TYPE OF EMPYEMA

TUBERCULOSIS	TOTAL	WORKING	LIVING	LOST	DEAD
Mixed infection	23	2 or 10%	6 or 26%		15 or 64%
Pure tuberculosis	48	6 or 13%	14 or 29%	2 or 4%	26 or 54%
Seropurulent	179	24 or 14%	49 or 27%	22 or 12%	84 or 47%
Total	250	32 or 13%	69 or 27%	24 or 10%	125 or 50%

CONCLUSIONS

1. Lack of any treatment carried a higher mortality than the treated groups in all types of effusions.

2. Empyema, both tuberculous and mixed with other pyogenic organisms, carried higher mortalities than did simple serous or seropurulent effusions.

3. The mortality rates were practically equal in purulent tuberculous empyemas and those with tuberculosis mixed with other pyogenic organisms. The number of the latter were about equal to the former.

4. In the mixed infection group, surgical treatment resulted in higher mortality in this series than did nonsurgical treatment, due to lack of surgical experience in the earlier years and the condition of the patients on admission.

5. In simple tuberculous empyema, surgical treatment reduced mortality more than did nonsurgical treatment, possibly because occurring since the advent of induced pneumothorax.

6. In serous and seropurulent effusions, the mortality was much reduced by surgical treatment.

7. Treatment of some sort in all types of pleural fluids affected favorably any underlying pulmonary tuberculosis, increasing the length of life.

8. Induced pneumothorax seemingly increased the incidence of pleural effusions but the pathologic condition following this etiologic factor seemed to be more amenable to treatment than that following other etiologic factors.

9. Mixed types excepted, so far as the future therapy is concerned, indications are that more favorable results will follow; first, aspiration, with or without instillation of some chemical; if this fails, some form of collapse therapy should be instituted along with this procedure. If that fails, resort should be made to more radical measures, such as thoracoplasty, or combination Schede operation, with possibly closed or open drainage. The interval of time given to each of these therapeutic measures should not be too protracted. When results are not being obtained, recourse should be had to any one of the other measures which may be indicated in that particular case, usually without delay.

10. Active treatment is indicated in removal of fluids to facilitate continuation of free pleural space and pneumothorax also to reduce the toxicity by removal of infected fluids and replacing with air and some antiseptic solution, possibly alcohol or gomenol.

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THE TREATMENT OF TUBERCULOUS EMPYEMA*†

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THE purpose of this communication is to report the clinical course and the end-results obtained in a series of 21 cases of toxic tuberculous empyema treated conservatively and followed up for periods ranging from three and one-half to eleven years. The clinical progress and the ultimate outcome of a case of tuberculous empyema depend not only upon the severity of the pleural infection, but largely also upon the extent and activity of the pulmonary tuberculous lesion which is practically always present. Secondary pyogenic infection of the pleural cavity, a bronchopleural fistula, or a draining sinus in the chest wall may complicate the case still further.

The principles underlying the treatment depend, to a large extent, upon how many of the complicating factors just mentioned are present in the case under consideration. The therapeutic measures commonly employed in tuberculous empyema may be conveniently classified as (a) conservative and (b) radical.

The conservative measures frequently used are: (1) aspiration, (2) aspiration and air replacement, (3) aspiration and irrigation with antiseptic solutions such as saline, gentian violet, acriflavin, potassium permanganate, etc., (4) oleothorax, with 5 to 10 per cent gomenol. Gentian violet in 0.1 per cent solution is the most frequently used for irrigation in cases complicated by a secondary pyogenic infection, and is said to be effective only against gram-positive organisms. Gomenol is used to combat the tuberculous infection, but so far as ultimate results are concerned, the reports published are as yet inconclusive.

The more radical measures employed are: (1) intercostal tube drainage by the open or "air-tight" method, (2) thoracotomy, (3) surgical collapse of the empyema cavity by major thoracoplastic surgery. Though intercostal drainage may be considered a minor surgical procedure, it is listed among the radical measures, because it is deemed a radical step to make an opening into a tuberculous pleural cavity which is almost certain to leave a draining sinus. Surgical drainage, either by the intercostal route or by rib resection, is usually instituted in the early acute stage of the disease for the relief of toxic symptoms, though intercostal tube drainage is sometimes employed later in the disease if fluid accumulates after a collapsing operation. It is fairly generally agreed that surgical drainage is contraindicated in a case of purely tuberculous in-

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†Received for publication, June 5, 1933.

fection, but it is frequently resorted to where a secondary pyogenic infection complicates a tuberculous empyema. Thoracoplastic collapsing operations are frequently employed in all types of tuberculous empyema if the purulent fluid persists, provided the patient is considered a fair risk for major surgery.

While it is generally conceded that conservative measures should be attempted first and that a cure may sometimes result from such measures, a review of the recent literature on the subject reveals that competent authorities employ conservative methods rather tentatively, mostly as preliminaries to major surgery, or as palliatives in patients unsuitable for thoracoplastic surgery. Thus Archibald[1] advises thoracoplasty in cases of seropurulent tuberculous effusions if the effusion persists after five to six aspirations, and immediate operation in cases with frank pus. Hedblom[2] advises thoracoplasty even in purely tuberculous empyema without evidence of pulmonary disease if the fluid persists from six months to one year. The prevailing opinion is reflected in the following conclusion of the Committee on Tuberculous Empyema of the American Sanatorium Association[3] which collected statistical data on the subject by the questionnaire method:

"From a study of the 235 cases, no doubt should exist in the minds of even the most skeptical, that, where the patient's condition warrants, no time should be lost in advising extrapleural thoracoplasty."

In view of this trend toward radical surgery in tuberculous empyema, the following report of the results obtained by a conservative method should be of interest.

REPORT OF CASES

This report is based on a series of 21 cases, 12 male and 9 female, of ages ranging from twenty-one to thirty-eight years, which were observed at the Rhode Island State Sanatorium for the last eleven years. Nineteen of these patients developed empyema during the course of treatment with induced pneumothorax. One was admitted with a spontaneous pyopneumothorax, and one was admitted with a tuberculous empyema of unknown origin. These 19 cases of toxic empyema complicating therapeutic pneumothorax, represent all the patients in whom this complication occurred in the institution between 1921 and 1931. Patients with seropurulent effusion who had no persistent toxic symptoms were not included. Though technically such cases may be said to suffer from tuberculous empyema, clinical experience shows that this type of empyema has no more effect on the outcome than serous effusions, and were treated as such. The 2 cases not complicating therapeutic pneumothorax were included because they were treated in the same manner, and the reaction to the treatment was similar to that of the other cases. Tubercle bacilli were found in the sputum of all these patients some time during the course of the disease.

All the patients who developed the empyema while under observation had a rather stormy onset, with fever ranging from 101° to 104°, and considerable prostration. Nine patients started with a serous effusion that turned purulent in three to five months. In 11 patients the effusion was purulent from the start. Perforation of the lung was definitely responsible for the empyema in 5 instances, but I am under the impression that there were more cases of perforation which were not recognized. Five patients had bronchopleural fistulas, and 3 had sinuses in the chest wall. Several cases had slight needletrack infections which did not break through and were therefore not of any particular significance.

Unfortunately, there is no satisfactory record of bacteriologic studies of the pus, so that it is impossible to state how many of these cases had a purely tuberculous infection and how many had mixed infections. From the severity of the symptoms, however, one would judge that mixed infection was present in a number of cases, especially in those who had perforation of the lung or bronchopleural fistulas.

TREATMENT

The treatment was uniform in all these cases. It consisted of aspiration of as much of the pus as possible, replacing it with air, and injection of 2 to 3 c.c. of a saturated alcoholic solution of methylene blue just before the needle was withdrawn. This treatment was repeated at varying intervals, depending upon the rapidity with which the fluid reaccumulated. The amount of air introduced was dependent upon the amount of fluid aspirated, and the intrapleural pressure. In large effusions with a free pleural cavity, a slightly negative pressure was left at the end of the treatment. If the pleural cavity became limited and further collapse of the lung was desired, enough air was introduced to produce a slightly positive pressure. This procedure was repeated periodically, as long as there was any fluid that could be aspirated, though the intervals between treatments could be gradually increased as the reaccumulation of the pus became slower. The successful cases remained under treatment for from eight months to four years. The average length of treatment was approximately two years.

CLINICAL COURSE

The clinical course varied in proportion to the severity of the pleural infection and to the extent and activity of the original pulmonary disease. In the majority of cases that terminated fatally, active and progressive pulmonary disease was present. The clinical picture of the surviving cases can be most clearly presented by considering them under three headings: first, as to relief of toxic symptoms; second, as to maintenance of lung collapse when indicated; third, as to ultimate reexpansion of the lung and obliteration of the empyema cavity.

Relief of Toxic Symptoms.—All the patients under consideration had considerable fever, prostration and other toxic symptoms at the time the

treatment was commenced, except for one patient who was admitted with an empyema of unknown origin. He was afebrile, but gave a history of fever lasting for several months, and at the time of admission was anemic, underweight and presented other symptoms of chronic infection. As a rule, evidence of improvement was manifested by lowering of fever and relief of other toxic symptoms after one or two treatments, but there were no spectacular results in any of these cases. Progress toward relief of all symptoms of toxemia was rather slow. Considering the fever as an index to toxemia, it took from five months to two years for the surviving patients to become persistently fever free. One patient who finally recovered, ran some fever for four years, but activity of the pulmonary lesion was probably at least partly responsible for the fever in this case. One patient who became fever free in June, 1927, had a relapse of fever to 101° in February, 1928, coincident with a recurrence of thick pus. The fever as well as the pus disappeared after about four months of treatment, but a similar relapse occurred in February, 1929, which cleared up in two months. The lung was then allowed to reexpand and the patient made an uneventful recovery. Another patient whose temperature became persistently normal in December, 1925, had a relapse of fever with a recurrence of sanguinopurulent fluid in April, 1927, the fever lasting for about six months.

Maintenance of Collapse.—In none of the patients in this series was a therapeutic collapse unfavorably affected by the empyema. In those patients in whom an unsatisfactory collapse was a factor in the unfavorable outcome, the collapse was ineffective at the time the empyema developed. In two of the successful cases the empyema, or rather the conditions responsible for the development of the empyema had an obviously favorable effect on the collapse. One of these two cases had an entirely ineffective collapse after six months of treatment. The pulmonary symptoms, such as frequent hemoptysis, etc., continued, and an upper lobe cavity remained unaffected. With occurrence of the effusion, the cavity promptly collapsed and the pulmonary symptoms subsided. This was probably due to a rupture of adhesions, causing an effusion and at the same time allowing the cavity to collapse. In another patient who had been under treatment with pneumothorax for two months, tubercle bacilli disappeared from the sputum immediately after the occurrence of perforation of the lung, resulting in a severely toxic pyopneumothorax. This patient finally recovered. Judging by repeated roentgenoscopy and sputum examinations, it seems that in three other cases with a collapse which was only partly effective, better collapse of cavities was gradually obtained by contraction of the visceral pleura and fibrosis caused by the empyema.

The collapse was maintained by replacing the purulent fluid with air, leaving a positive pressure in such cases where a tendency of premature

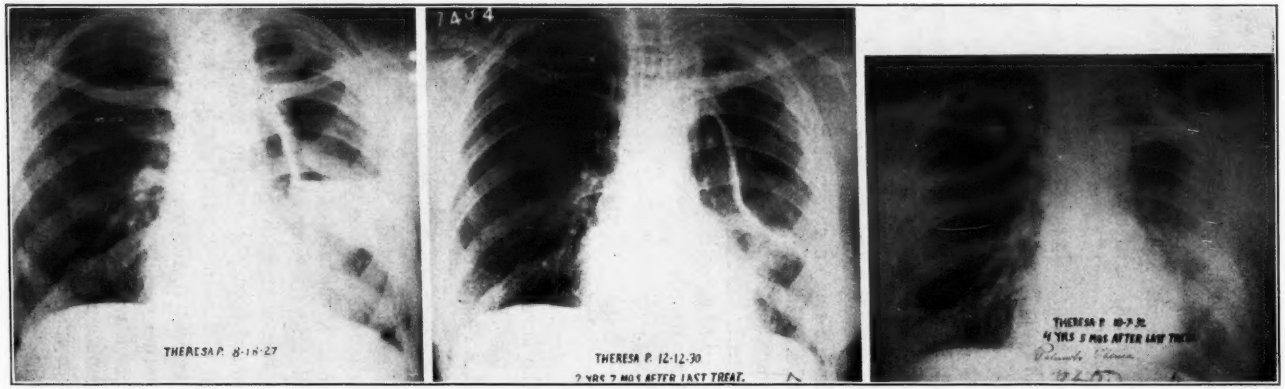

Fig. 1. Fig. 2. Fig. 3.

Fig. 1.—Case of T. P., film taken Aug. 18, 1927, about three years after the development of the empyema. Note reexpansion of the lower lobe with limited pyopneumothorax.

Fig. 2.—Same case as shown in Fig. 1, film taken Sept. 12, 1930, two years and seven months after the last aspiration and refill. Note absorption of residual effusion, limited pneumothorax and thickened pleura. (Courtesy of Dr. J. I. Pinckney.)

Fig. 3.—Same case as Figs. 1 and 2, film taken Oct. 7, 1932, showing reexpansion of lung.

reexpansion was noted. But pressure above 10 cm. of water was rarely used. In four patients refills could be continued after the pleural cavity was entirely freed of the effusion. In the others, the pleural space gradually became so limited that only a small pocket of pyopneumothorax remained and continuation of refills was impossible. But this process occurred so gradually, that no reactivation of the pulmonary lesion or opening of cavities resulted.

Ultimate Reexpansion of the Lung.—The empyema cavity was finally obliterated and the lung reexpanded in all the 11 patients who recovered, but the time required for the reexpansion to be accomplished and the condition of the reexpanded lung varied considerably. In four cases a large pneumothorax pocket was left after the empyema cleared up entirely, and pneumothorax refills were continued for more than three

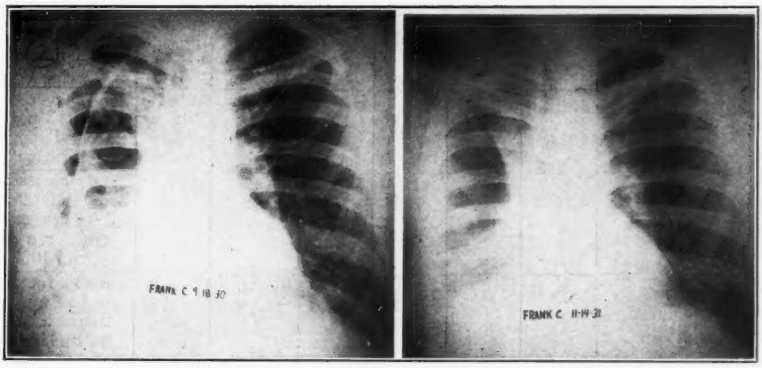

Fig. 4. Fig. 5.

Fig. 4.—Case of F. C., film taken Sept. 18, 1930. Lung in process of reexpansion after clearing up of an empyema that recurred twice in three years. Note thickened pleura.

Fig. 5.—Same case as Fig. 4, film taken Nov. 14, 1931. Lung reexpanded with some retraction of the upper lobe.

years in one case, and from two to five months in the other three cases. In these cases reexpansion did not differ much from that observed in uncomplicated cases of pneumothorax which are allowed to reexpand, except that the process was much slower, and there was more retraction of the chest wall and moderate shifting of the mediastinum. In the other patients, partial reexpansion began after two or three months of treatment, and continued gradually, the pyopneumothorax pocket growing progressively smaller. In these cases, expansion usually started at the base, resulting in adhesions obliterating the lower part of the pleural cavity. This process usually continued until only a small area of pyopneumothorax limited to the outer part of the axillary region remained. In several cases the treatment was abandoned at this stage, but in all

the survivors the small amount of pus and air finally disappeared, though in one case the lung did not completely reexpand until four years after the treatment was discontinued. It is impossible to state how long the residual small pocket of pyopneumothorax persisted in each case, as

TABLE I

RÉSUMÉ OF LIVING PATIENTS

CASE	PNEUMOTHORAX INDUCED	SEROUS FLUID	PUS	LAST ASPIRATION	LAST REFILL	REEXPANSION	PRESENT CONDITION	REMARKS
A. H. m. 21	Jan. 7, 1922	July, 1922	Sept., 1922	May, 1923	March, 1927	Good	Well, working	Effusion improved the collapse
E. H. f. 37	Oct. 24, 1922	0	Jan., 1923	Nov., 1924	April, 1925	Poor, fibrosis	Well, doing housework	
F. G. m. 34	Spontaneous April, 1924	0	April, 1924	May, 1927	May, 1927	Poor, marked fibrosis	Well, working	Had a fair-sized bronchial fistula
T. P. f. 28	May 13, 1924	0	July, 1924	May, 1928	May, 1928	Good	Well, doing housework	Empyema resulting from lung perforation
N. M. f. 34	Nov. 5, 1924	March, 1925	June, 1925	July, 1928	July, 1928	Fair	Well, doing housework	Fluid cleared up March, 1926, recurred April, 1927
I. S. f. 29	Feb. 3, 1925	0	Nov., 1925	Jan., 1928	Jan., 1928	Poor, fibrosis	Well, doing housework	Small intermittent bronchial fistula — also small chest sinus —healed since 1929
F. B. m. 24	July, 1925	Nov., 1925	April, 1926	May, 1927	May, 1927	Fair	Well, working	
F. C. m. 20	July 6, 1926	0	Jan., 1927	Feb., 1930	July, 1930	Good	Well, working	Relapse of fever and pus Feb., 1928, and again Feb., 1929
J. B. f. 24	March 26, 1927	Sept., 1927	Nov., 1927	July, 1928	July, 1928	Good	Well, working	
J. S. f. 21	Aug. 27, 1929	Sept., 1929	Jan., 1930	July, 1931	Sept., 1931	Good	Well	
B. W. m. 26	Idiopathic empyema Sept., 1929	0	Sept., 1929	May, 1930	May, 1930	Good	Under treatment	Developed lesion in opposite side — treated with pneumothorax

roentgenologic examinations were not made frequently enough, but four years is probably the longest period.

The condition of the reexpanded lung apparently depends to a considerable extent upon the original pulmonary lesion. In six cases, including 3 patients in whom reexpansion was optional, there was a fairly good reexpanded lung, though there was some evidence of fibrosis in the upper lobe and at the base. In the remainder of the cases, there was considerable fibrosis and retraction.

END-RESULTS

Of the 21 cases considered, 11 (or 52 per cent) are cured, and 10 (or 48 per cent) are dead. Of the 11 cured patients, 10 are entirely free of symptoms and are able to work, and one is under treatment with pneumothorax for a newly developed lesion on the opposite side, but the empyema has entirely cleared up and the lung reexpanded. Among those who recovered, are included 2 patients whose empyema developed as a result of perforation of the lung, and of these 2, one had also a fair-sized bronchopleural fistula. One patient with a small draining sinus in the chest wall and a small intermittently patent bronchopleural fistula also recovered. In this case, the sinus persisted for about two years and has been healed for the last three years. Of the 10 patients who died, four showed considerable improvement temporarily, and the ultimate fatal outcome was due to the progressive pulmonary disease. Six failed to show improvement.

The empyema was probably an important factor in the unfavorable outcome of 3 cases. In 2 of these, large bronchopleural fistulas were present.

DISCUSSION

Considering the fact that only patients with prolonged symptoms of toxemia were included in the group just described, the end-results obtained are rather gratifying. These results compare favorably with those reported by other writers. Thus Hayes[4] reports 32 cases treated by various methods, with 13 living and only one able to work. Archibald[1] reports 5 recoveries out of 19 cases subjected to thoracoplasty. Berry[5] obtained 15 cures out of 75 cases of tuberculous empyema with mixed infection. Hedblom,[6] on the other hand, reports an exceptionally large percentage of recoveries as a result of thoracoplasty. He obtained a cure in 74.7 per cent in a group of 23 patients of purely tuberculous empyema, and an obliteration of the empyema cavity in 64.4 per cent in a series of 59 cases with mixed infection, while he was able to effect a cure in only about 10 per cent in a group of 61 patients treated by minor surgical methods only. Somewhat similar figures were reported by various workers in reply to a questionnaire sent out by the Committee on Tuberculous Empyema of the American Sanatorium Association. Of

134 cases of virulent tuberculous empyema treated by thoracoplasty, 65 (49 per cent) were reported as cured, and 28 (21 per cent) as "clinically well."

Considered from a purely statistical point of view, these figures certainly constitute a strong argument in favor of early thoracoplasty. It must be borne in mind, however, that only patients with a good contralateral lung who are in reasonably good general condition are selected for major surgery, while conservative methods are used in a large number of hopeless cases with extensive bilateral pulmonary disease. The percentage of recoveries as a result of major surgery may serve as an index of the conservatism in the selection of cases, and the technical skill of the surgeon, but it can hardly be considered a fair basis of comparison in passing judgment upon the merits of the different methods of treatment. At any rate, in my own series of cases, the results of conservative treatment seem to be satisfactory. Of course the particular method used may be an essential factor in bringing about these results.

The Effect of Methylene Blue.—The first two cases treated with methylene blue in the manner described above were reported by Stivelman and myself[7] in 1921. The idea occurred to us after we injected a small amount of methylene blue into the chest of a patient suffering from tuberculous empyema, in order to determine the presence of a bronchial fistula. As the patient showed definite though temporary improvement, we decided to try it on other patients. A rational basis for the use of methylene blue is provided by the experiments of Lewis and DeWitt.[8] They showed that dilute solutions of this dye are capable of inhibiting the growth of tubercle bacilli, and that the blood of dogs that had been injected with methylene blue also is bacteriostatic against the tubercle bacilli. These experiments suggest that this dye is effective in the presence of organic matter. Nobecourt[9] and his coworkers showed that methylene blue is also effective in pyogenic empyema following bronchopneumonia.

The amount used (about 3 c.c. of a saturated alcoholic solution) seems rather small, but in the first cases treated a febrile reaction followed each injection. We hesitated, therefore, to increase the amount. Large amounts of methylene blue have been used in Europe, and Rosenstein[10] reports a fatality following the injection of 150 c.c. of a 5 per cent aqueous solution of this dye. Of course, with small amounts, as used by us, the toxic effects need not be taken into consideration. It is, of course, impossible to state with any degree of certainty how much the dye was responsible for the results obtained, but there is no doubt but that it helped to relieve the toxic symptoms and to slow down the reaccumulation of pus. It probably also facilitated gradual pleural agglutination, thus reducing the size of the empyema cavity.

It is needless to state that it is not my intention to recommend this treatment in all cases of tuberculous empyema. If an extremely virulent

pyogenic infection complicates a tuberculous empyema, surgical drainage may be indicated, but it seems to me that some conservative measure should be given a thorough trial in all cases where the infection is predominantly tuberculous, even if a secondary pyogenic infection is present. While there is no record of bacteriologic studies in the series of cases above described, there is no doubt but that those patients who had perforation of the lung and those with bronchopleural fistula, had secondary infections; yet four cases of this type recovered under conservative treatment. Furthermore, Danna[11] has shown that purely pyogenic empyema can be treated successfully with aspiration and air replacement. Whether or not this method of treatment is preferred in nontuberculous cases of empyema, it should certainly be the method of choice in a tuberculous individual where surgical drainage may be disastrous.

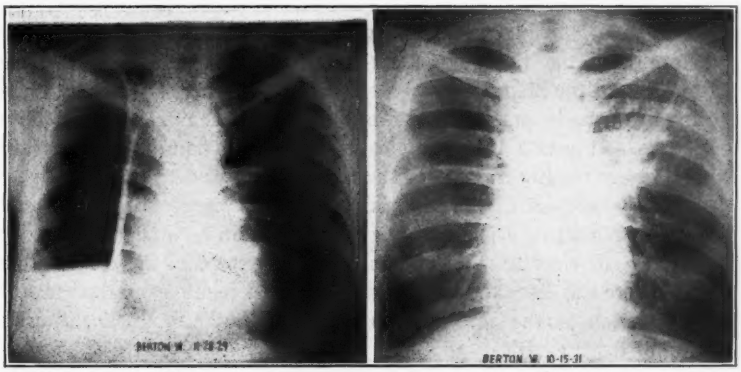

Fig. 6. Fig. 7.

Fig. 6.—Case of B. W., film taken Nov. 28, 1929, showing pyopneumothorax. Note thickening of pleura.

Fig. 7.—Same case as Fig. 6, film taken Oct. 15, 1931, showing reexpansion of the right lung. Note newly developed lesion in the left upper lobe. (Patient doing well under pneumothorax treatment on left.)

As to thoracoplasty, there are certain types of empyema where the indication for surgical collapse is obvious. If there is an active pulmonary lesion on the side of the empyema, surgical collapse may, of course, be indicated for the pulmonary condition, and the empyema is then only of secondary consideration. Cases with persistently draining sinuses, sometimes produced by needless surgical drainage, require plastic operations for the collapse of the empyema cavity. Large bronchial fistulas may sometimes be closed by thoracoplastic surgery. It is doubtful, however, whether major surgery is indicated in the majority of cases with closed empyema.

It is claimed that after purulent fluid has persisted in the pleural cavity for a long time, the lung cannot reexpand and the empyema cavity cannot, therefore, be obliterated without surgery. It seems, how-

ever, that with persistent aspiration and air replacement, the lung will ultimately reexpand to a considerable extent and the empyema cavity will be obliterated. Some retraction of the chest wall, shifting of the mediastinum, and elevation of the diaphragm contribute to the obliteration of the empyema cavity where the lung is not completely reexpanded. In none of my cases was the shifting of the mediastinum sufficient to cause any discomfort.

It has been stated that tuberculous empyema interferes with therapeutic collapse of the lung because of its tendency to produce an obliterative pleuritis, thus causing reopening of collapsed cavities and reactivation of the disease. In my experience, this situation occurred more often in patients with serous or mild seropurulent effusion. These effusions show a tendency to rapid absorption, resulting in a premature reexpansion of the lung and formation of adhesions. In the more toxic empyema, however, the absorption of the effusion is very slow, and the resulting obliteration of the pleural space occurs so gradually, that it does not interfere with the therapeutic effect of the collapse. Moreover, the obliteration of the pleural cavity almost always begins at the base, where the lung is usually normal, and the upper lobe remains collapsed for a long time. It seems that the elastic normal portion of the lung reexpands more readily in spite of the thickened visceral pleura, while the plastic diseased part of the lung is prevented from reexpansion by the firm, thick pleura. Ultimately, however, a certain amount of expansion takes place, depending more or less upon the extent of the original disease. Where the disease was extensive, the final result is marked fibrosis and retraction of the chest wall, not unlike that seen in spontaneous healing of large cavities.

SUMMARY AND CONCLUSIONS

1. Twenty-one cases of tuberculous empyema treated with aspiration, air replacement and injection of methylene blue, have been reported.

2. Eleven patients including 2 cases with spontaneous pneumothorax, and 2 with bronchopleural fistulas have recovered.

3. Small amounts of methylene blue are apparently of value in the treatment of tuberculous empyema.

4. Surgical drainage, while indicated in exceptional cases of virulent pyogenic empyema, is unwarranted in cases with predominating tuberculous empyema, even if a mixed infection is present.

5. Thoracoplasty is indicated in cases with active lung disease on the empyema side and in open empyema, but many patients with closed empyema may recover without major surgery.

6. Reexpansion of the lung and obliteration of the empyema cavity can occur even in cases where an empyema persisted for a long time.

7. Toxic empyema does not usually interfere with therapeutic collapse, and in some cases it may have a favorable effect on the collapse.

My thanks are due to Drs. Mary M. Hamblet and A. A. Karan of the medical staff and Mr. Aram Haytaian of the laboratory staff of the Rhode Island State Sanatorium for valuable aid in the preparation of this paper.

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Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

Selected Abstracts

George L. Waldbott. So-Called Thymic Death. VI. The Pathologic Process in Thirty-Four Cases. Am. J. Dis. Child. **47:** 41, 1934.

This important work concerns 132 cases in which the diagnosis of status thymicolymphaticus was made by a number of outstanding pathologists. Thirty-four cases in which no adequate cause of death had been determined were selected for special study.

"In eleven of the thirty-four cases death occurred without previous illness, nor were there any pathologic lesions to indicate the presence of a previous illness. *In seven a syndrome developed manifesting dyspnea, stridor, fever and shock.*

"The lungs of all these patients presented uniform changes, characterized by capillary congestion, extravasation of blood cells and edematous fluid. These lesions alternated with areas of emphysema and atelectasis."

In seventeen cases petechial hemorrhages were present involving the heart, pleura, lymph glands and various other viscera. In some, there were dilatation of the right side of the heart and degenerative changes in the liver and edema and capillary congestion in other organs than the lungs. Hypoplasia of the suprarenal glands and hyperplasia of the lymphoid organs, as noted by other authors, were present.

Comparison of the above mentioned changes with those reported in anaphylactic shock in man revealed almost a complete identity of the two conditions. "Eosinophilia of the tissues and an allergic family or personal history point further to this conception."

It is thus believed that so-called "thymic death" may be the result of a primary anaphylactic edema of the lungs and ensuing asphyxiation. To uphold such a theory it is necessary, as is pointed out, to assume that anaphylactic shock may occur from incorporation into the body of nonprotein substances; also that absorption of shock-producing antigen may take place by ways other than by injections.

Harry C. Ballon.

George L. Waldbott and G. E. Anthony. So-Called Hyperplasia. IV. A Follow-Up Study of Thirty Cases. Am. J. Dis. Child. **47:** 34, 1934.

In this study Waldbott and Anthony found that a follow-up study in thirty children whose condition had been diagnosed by the x-ray method as "enlarged thymus" revealed that twenty-four were afflicted with an allergic disease as evidenced by the history and by skin tests. In four of the remaining cases such a diagnosis was questionable. In one, such marked sensitivity was encountered that a scratch test with walnut produced a definite anaphylactic reaction, although the child had never eaten walnut. The possible relationship of thymic enlargement to anaphylactic death is discussed, and thorough allergic studies in cases with x-ray evidence of enlarged thymus are suggested.

Harry C. Ballon.

Lope de Carvalho and Egas Moniz. The Visibility of the Pulmonary Vessels. Acta Radiol. **14:** 433, 1933.

The authors report the first successful visualization of the pulmonary vessels in the living human being. After experiments on rabbits, dogs, and monkeys, they arrived at a method of injecting concentrated sodium iodide solution (120 per cent) into the right auricle by means of a radiopaque catheter introduced into the cubital vein of the arm and advanced until it was seen to be in the right auricle. Clotting within the catheter during its passage through the veins was prevented by injection of 10 per cent solution of sodium citrate.

With the end of the tube in the right auricle the patient was placed against the x-ray film, and x-ray exposure was made immediately following injection of from 6 to 8 cm. of the concentrated sodium iodide solution.

The authors report that the outstanding symptoms experienced by the patients were slight headache and brief spell of coughing. They also mention signs of iodism to which they apparently attach little importance. In forty-eight cases of pulmonary tuberculosis examined, in which the patients were subjected to this procedure, they could find no signs of aggravation of the disease. In fact, they mention the possibility of improvement of their patients as a result of this test as shown by the change from a subfebrile state to normal temperature and increase in weight.

The authors are quite vague in stating the advantages to be gained in diagnosis by the use of this new method. They state that "the fact that the condition of the pulmonary vessels can be ascertained without any harm to the patient would constitute in itself a great advance in radiological semeiotics." In addition they mention the advantages of

obtaining pictures of the lung roots, the tuberculous lesions of the lung, and the state of pulmonary circulation in patients subjected to treatment by artificial pneumothorax. However, they do not state just what additional diagnostic facts are gained from these observations. They do point out that the angiopneumographs show that the normal pulmonary design is due to the vessels and not to the bronchi, an observation generally accepted before the introduction of this method. An important observation which they point out is the slowness of the flow of blood in the collapsed lung, a point on which there has been some controversy.

Altogether the thesis strikes one as being hazardous out of all proportion to the information which might be gained through its use. It may be granted that the method is a useful one for animal experimentation but hardly to be considered for diagnostic use in the human being.

Joseph Weinberg.

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The American Association for Thoracic Surgery

Boston, Mass., May 31, June 1 and 2, 1934

Hotel Headquarters—Copley Plaza Hotel, Copley Square

Program

Thursday, May 31

9:00 a.m. *Building C. Amphitheatre, Harvard Medical School, Longwood Avenue.*

1. Business Meeting of the Association.
2. A Consideration of the Dangers of Lobectomy in Relation to the Technic of the Operation.
   Dr. Edward Archibald, Montreal.
3. Lobectomy in Children for Bronchiectasis.
   Dr. J. V. Bohrer, New York.
4. Lobectomy Left Upper Lobe for Carcinoma.
   Dr. Carl Eggers, New York.
5. The Effect of Sudden Occlusion of Either Primary Branch of the Pulmonary Artery on the Cardiac Output and Pulmonary Expansion in Dogs.
   Dr. Richmond L. Moore, Dr. Humphreys, and Dr. Cochran, New York.
6. Vascular Changes in Experimental Atelectasis, Morphological, Physiological and Biochemical.
   Dr. William E. Adams, Chicago.

7. The Pleural and Pulmonary Lesions Resulting From the Intrapleural Injection of Mycobacterium Tuberculosis (bovis) in Rabbits.
   Dr. Willis S. Lemon, Rochester.

12:45 p.m.

Luncheon, Vanderbilt Hall, Corner of Longwood Avenue and Louis Pasteur Avenue.

2:00 to 5:00 p.m. Building C. Amphitheatre, Harvard Medical School, Longwood Avenue.

1. Some Reflections on Intrathoracic Malignant Disease.
   Mr. A. Tudor Edwards, London, England. (By special invitation.)
2. Classification of Primary Bronchogenic Carcinoma in Relation to Treatment and Prognosis.
   Dr. William Tuttle, Dr. Nathan Womack, and Dr. Evarts A. Graham, St. Louis.
3. A Topographical Classification of Carcinoma of the Lung. Its Clinical Application in the Operative Treatment.
   Dr. Harold Neuhof, New York City.
4. Bronchial Carcinoma, Diagnosis by Bronchoscopic Biopsy in a Series of 50 Cases.
   Dr. Chevalier L. Jackson, Philadelphia.
5. Intrathoracic Neurofibroma.
   Dr. Stuart W. Harrington, Rochester, Minn.
6. Incidence of Hiatus Hernia in Pregnant Women and Its Significance.
   Dr. Leo Rigler, Minneapolis.

5:00 to 6:00 p.m.

Tea with Dr. Elliot C. Cutler, Laboratory of Surgical Research, Harvard Medical School, Building C.

Friday, June 1

9:00 a.m. to 12:30 p.m. Building C, Harvard Medical School, Longwood Avenue.

1. Executive Meeting of the Association.
2. Thoracoplasty in Bilateral Cavernous Tuberculosis.
   Dr. Harold Jessen, Davos-Platz, Switzerland. (By special invitation.)
3. Simultaneous Bilateral Artificial Pneumothorax in the Treatment of Pulmonary Tuberculosis.
   Dr. Thomas Kinsella, and Dr. P. M. Mattill, Oak Grove, Minn.
4. The Importance of Resection of the Transverse Processes as a Primary Procedure in the Obliteration of Paravertebral Cavities and in the Revision of an Inadequate Thoracoplasty.
   Dr. Emile Holman, San Francisco.
5. Scaleniotomy With or Subsequent to Phrenicectomy in the Treatment of Pulmonary Tuberculosis.
   Dr. Lincoln Fisher, Waverley Hills, Ky.
6. The Results of Operations on the Phrenic Nerve.
   Dr. Adrian V. S. Lambert, New York.
7. The End-Results in the Surgical Treatment of Pulmonary Tuberculosis.
   Dr. Carl Hedblom, Chicago.

12:45 p.m.

Luncheon, Vanderbilt Hall.

2:00 p.m. to 5:30 p.m.

Demonstrations of research problems under investigation in the various laboratories of the Harvard Medical School and local hospitals.

7:00 p.m. Aesculapian Room, Harvard Club, Boston.

PRESIDENT'S DINNER.

PRESIDENT'S ADDRESS.

Dr. George Heuer.

Saturday, June 2

8:30 a.m.

Operative clinics and demonstrations at the Deaconess Hospital, Massachusetts General Hospital, and Peter Bent Brigham Hospital.

THE FOLLOWING PAPERS ARE TO BE READ BY TITLE:

1. Dr. C. I. Allen, Detroit.
   A Case of Primary Carcinoma of the Lung. Report Four Years After Lobectomy.
2. Dr. David Ballon, Montreal.
   Bronchoscopy in Pulmonary Tuberculosis.
3. Dr. Harry Ballon, Montreal.
   The Clinical Significant Irregularities of the Diaphragm.
4. Dr. Maurice Berck, New York.
   Radiotherapy for Bronchorrhea in Bronchiectasis.
5. Dr. Norman Bethune, Montreal.
   "Pleural Poudrage," The Production of Pleural Adhesions as a Preliminary to Lobectomy.
6. Dr. Harold Beye, Iowa City.
   Multiple Empyemata, With Special Reference to Etiology and Localization.
7. Dr. J. Dewey Bisgard, Omaha.
   Experimental Thoracogenic Scoliosis.
8. Dr. E. F. Butler, Elmira.
   Postpneumonic Atelectasis as a Possible Cause of Chronic Empyema.
9. Dr. Pol N. Coryllos, New York.
   Thoracoplasty Versus Pneumothorax.
10. Dr. Elliot Cutler and Dr. Cornelius B. Wood, Boston.
    The Effect of Bronchial Occlusion.
11. Dr. H. Ryerson Decker, Pittsburgh.
    Primary Malignant Tumors of the Thymus Gland, With Report of Two Cases.
12. Dr. Max Einhorn, Dr. DeWitt Stetten, and Dr. William H. Stewart, New York.
    Report of a Case of Cardiospasm Due to a Diaphragmatic Gastric Hernia With Ulcer.
13. Dr. William A. Hudson, Detroit.
    Thoracoplasty, a Critical Study of Some Unsatisfactory Results.
14. Dr. Urban Maes and Dr. J. Ross Veal, New Orleans.
    The Mortality of Empyema. An Analysis of 75 Cases.
15. Dr. Harlan F. Newton, Boston.
    Spinal Anesthesia in Thoracoplastic Operations for Pulmonary Tuberculosis.

16. Dr. E. J. O'Brien, Detroit.
    Thoracoplasty.
17. Dr. Richard Overholt, Boston.
    Pneumonectomy for Carcinoma of the Lung. A Report of the Successful Removal of the Right Lung for Carcinoma.
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